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ETIOLOGY AND TREATMENT OF CERTAIN KINDS OF COUGH.

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Cough is one of those symptoms we are called upon constantly to treat. At times the diagnosis of its cause is relatively easy, and our treatment satisfactory. It may last, it is true, for some days despite our efforts to relieve, and during this time cause moderate annoyance, or even considerable distress. Still, after a fair trial of remedies judiciously employed a measure of benefit is obtained, and both patient and physician are hopeful as to a speedy cure, and both are tolerably satisfied with the amount of success accomplished in a given period. Again, there are cases in which we know from the first that whatever treatment may be followed the obstinate cough, in the nature of things, must persist, and arrest, except from increasing doses of anodynes, can rarely be effected. Such cases we are familiar with in certain forms of pulmonary and laryngeal phthisis.

There are other kinds of cough which are also met with quite frequently; yet their diagnosis is made with difficulty, and their treatment, despite repeated changes, fails to accomplish much in the way of abatement and cure. This is true not only of the patients who go first to the family physician in search of help, but also of those who in the beginning of their trouble gravitate toward some prominent specialist.

In the class of cases where the general practitioner is usually at fault I would place the cough which is under dependence of an engorged lingual tonsil. On at least two occasions I have treated wives of prominent medical practitioners who were sufferers from annoying symptoms

[1] Read before the Climatological Association, June 14, 1895.

of this origin, although previous to my seeing and treating them the nature of the trouble had not been recognized. In these cases there was no chest affection and no apparent throat trouble sufficient to cause the distressing cough, or other symptoms. There was no evident local disease elsewhere in one case; in the other there were joint symptoms of rheumatic gout. In one case the cough had resisted many usual remedies given internally, and the repeated employment of sprays and inhalation of balsamic vapors.

The cough in these cases is frequent, dry, paroxysmal. Anodynes, even in moderately large doses, fail to afford relief. At times the cough is occasioned by the sensation of a foreign body lodged at the base of the tongue, like a bristle, a bread-crumb, or a bit of meat, but it is impossible to dislodge anything or get rid of the annoying sensation.

Accompanying the cough there may be a continuous desire to swallow constantly, and the effort of deglutition may be performed with some difficulty. Indeed, I have had one case under my care in which the difficulty of swallowing was so great as to excite much apprehension lest choking should occur, and the young woman soon lost flesh and strength to a marked degree, through dread of taking her meals. With the difficulty of swallowing there may be a feeling of a constricting hand around the throat, which occasionally seems as if it would throttle the patient. This sensation is greatly increased when the patient lies down at night, and of course increases his terror.

But these are very exaggerated cases, and frequently nothing betrays the evidence of local irritation of the larynx from lymphoid hypertrophy at the base of the tongue, except an almost continuous cough. I have known such cases to be regarded for some time as phthisical, and again as hysterical.

When the obstinate cough is thought to be evidence of incipient phthisis, change of air, absence from business or household cares, cod-liver oil, and creosote begin to loom up as the only remaining means of helpfulness. If the patient be supposed to suffer from hysteria—and how often is the so-called "globus hystericus" made to account for what is caused by pressure from an offending mass—little or no treatment is insisted upon. The patient is often spoken of as an imaginary sufferer, for whom a cold douche, valerian pills, and some moral education sum up about everything which can be done.[1]

When these cases are examined with the laryngoscope, and it is only with the laryngoscope that they can surely be made out, we note the following conditions: The epiglottidean fossa, *i. e.*, the fossa between the epiglottis and the base of the tongue, is more or less completely filled

[1] I am more inclined to the belief than ever that the nervous cough of adolescents described graphically by the late Sir Andrew Clarke was simply a cough caused by an enlarged lingual tonsil.

up and distended by a slightly irregular but rounded mass of lymphoid tissue. This mass is sometimes deep red, sometimes pink, and again pale in color. It is often covered with irregular cauliflower excrescences not larger than a very small pea. Again, it is relatively smooth and glistening like certain forms of enlarged faucial tonsils in children. The mass may simply fill the fossa, pressing against the entire anterior surface of the epiglottis; or it may be so much larger on one side than it is on the other that the pressure on the epiglottis is only partial, and on the opposite side to the one where this is evident the fossa is not wholly filled up. Frequently the free border of the epiglottis is, to a more or less considerable extent, caught in or covered by the overtopping tonsillar mass; and it would seem to be particularly this portion of the mass which occasions the troublesome cough. When the patient phonates the mass occasionally separates from the free margin of the epiglottis; occasionally it shows no separation at all, but adheres under vocal efforts closely to it.

Frequently there are quite large veins distinctly defined on the tonsillar mass, and not seldom these veins will burst and allow more or less blood to come into the mouth and be expectorated, which I have known to cause the liveliest apprehension on the part of the physician and the patient. Fortunately, the bleeding soon stops, and the patient is none the worse for it, except mentally.

These enlargements of the lingual tonsil are uncommon among young children; they are also infrequent among young men and women; but toward middle life, in men and women, I have had numerous cases—more among women than men.

The causes of the enlargement are certain menstrual derangements, continued constipation, and an underlying rheumatic or gouty state. No doubt, micro-organisms may infect as readily, perhaps even more easily, this tonsillar mass than they do those masses at the faucial entrance.

In rare cases syphilis has doubtless localized itself in this region, either causing marked hyperæmia or a congestive condition, upon which a mucous patch may readily develop, as it does upon the faucial tonsils.

How should we treat this engorged lingual tonsil? Internally, we must give the salicylates in fairly large doses, and usually we shall obtain from their use very evident benefit. It is not essential in giving the salicylates to be able to discover some other manifest rheumatic symptoms; nor, indeed, should we feel compelled to obtain a clearly rheumatic history. Despite the absence of either the one or the other, we often get good results from this treatment.

In prescribing salicylic acid or the salicylates, it is very important to get salicylic acid obtained from the proper chemical source. That made directly from the oil of wintergreen is the only one which is safe

and judicious to use. The other is very apt to cause pain and nausea, or other symptoms of stomachal intolerance. While I believe sprays of some benefit, especially those of carbolic acid combined with the essential oils and boric acid, still these will not cure by themselves the lingual hypertrophy. Local applications of a stronger kind are necessary. Among these, I place foremost the galvano-cautery and compound tincture of iodine.

Excision of the tonsil by a specially devised knife or guillotine (Chappell's) has been recommended highly by a few prominent throat specialists, but thus far has not commanded general favor. The site of the disease makes it awkward for operation with the guillotine unless it be imperatively required, and the risk from annoying bleeding, or some other accident following excision, is not, in my judgment, as small as has been affirmed. Formerly, I treated these cases with repeated applications of the galvano-cautery, and, upon the whole, my results were gratifying; still, owing to the soreness and swelling which lasted for several days subsequent to the use of the cautery, I had reason occasionally to be troubled in mind.

I do not remember to have had an abscess from the peritonsillar structure after cauterization, but I know that several times the tonsil was so much inflamed that I sought relief for my patient through repeated lancing with a curved knife.[1] The great objection, however, to the use of the cautery in this region is the risk of burning the epiglottis, and particularly its free border. Unless the patient is phlegmatic and obedient, and holds himself very steadily, we may inadvertently produce an ugly sore which will give any amount of trouble before it heals.

Latterly, by repeated applications of compound tincture of iodine to the tonsillar mass with a curved brush or sponge-holder, and by the use of the salicylates internally, I have been able in a few weeks to reduce these enlarged tonsils so that they ceased to occasion cough or other symptoms of local distress. The applications of iodine may be repeated daily with considerable advantage, or as frequently as can be made without causing marked local soreness. Even when the cough disappears, or the obstructed deglutition is no longer present, the voice may be more or less hoarse and discordant for some time.

In using the galvano-cautery it must always be borne in mind that an unfortunate burning of the margin of the epiglottis may bring on a cough even more troublesome than the one we are trying to cure, and for this reason, after considerable experience, I am inclined to reserve its use for those cases in which internal treatment and the local use of compound tincture of iodine remain without curative effect.

[1] I have had two cases under my care in whom an abscess formed in this tonsil, and after causing great distress, *i. e.*, dyspnœa and choking, burst spontaneously, to the great and immediate relief of the patients.

Another form of cough occurs in young children, and is often ignored, or, if not ignored, the treatment is at least ineffectual, as it does not reach the cause of it. Frequently children cough repeatedly, and at night especially, on account of one of two conditions: either there is a dropping of thick mucus, or muco-pus, from the nasopharynx upon or into the larynx, or there is an irritation of the posterior turbinated bodies brought on by local congestion. The first condition is made evident frequently by the examination of the pharynx with an ordinary tongue spatula. So soon as the tongue is moderately depressed the child has an effort of gagging, and a large mass of mucus is seen between the free border of the palate and the pharyngeal wall, squeezed downward by the forced effort which just precedes its appearance. Usually this condition in children is due to more or less development of the pharyngeal tonsil or lymphoid tissue at the vault of the pharynx. It can be cured by a moderate scraping with the finger-nail of the right index-finger introduced behind and above the soft palate. If the finger be properly protected by a thick rubber nipple (*i. e.*, such a one as is used to cover the mouth of a nursing bottle) it will not be wounded by the child's teeth. No anæsthesia is required. The pain from the scraping is very slight, and the operation lasts but a few moments. To be thorough, two or more scrapings should be made at the time, or if the child is very restive after the first operation further interference may be delayed until a later and more favorable occasion. In some of these cases there is quite an amount of bleeding for a few moments during and immediately after the operation; but in my experience it has quickly ceased. If it were to continue I would advise swabbing the post-nasal space with a little of Mackenzie's tanno-gallic powder (three parts of tannin and one part of gallic acid). Indeed, I have made this application on more than one occasion as a simple matter of precaution, and with obviously a satisfactory astringent effect.

In the event of the hemorrhage being at all abundant or continuing for any length of time, the proper thing to do would be to place a plug of iodoform or sterilized gauze in the post-nasal space with the finger or a pair of post-nasal forceps, allowing a string to remain attached, so that the tampon could be removed at any moment it seemed advisable to do so.

For a few days subsequent to the scraping it is wise to spray the nasal and post-nasal passages with a mild antiseptic spray composed in part of carbolic or boric acid.

Sometimes there is really no adenoid tissue in the post-nasal space to account for the obstinate cough, and there is practically no hypersecretion of mucus or muco-pus from this region.

The nasal passages may be either tolerably pervious, or they may be notably occluded. Sometimes the occlusion is but little noticed in the day-time, but at night it becomes greatly aggravated, and especially

When the patient is lying on his back, he is restless and uncomfortable, throws himself about in bed, coughs frequently, and yet apparently there is not sufficient evidence in an ordinary inspection of the fauces and pharynx to account for these morbid phenomena.

At times the cough is relieved very much for some time by a suitably formulated nasal spray or a few applications of moderate severity to the nasal mucous membrane.

I have found albolene with camphor and carbolic acid one of the best combinations as a spray or vapor, and applications of carbolic acid and glycerin (from 1 part to 8 to equal parts of each ingredient) as the most useful local application by means of a nasal carrier, I have hitherto employed in these cases.

Whenever the cough is not altogether relieved by these means used in the manner referred to, I find it is most useful to paint over the posterior end of the turbinated bodies (as much as I am able), and also the vault of the pharynx, with carbolic acid and glycerin (1 part carbolic acid to 6 or 8 parts of glycerin). In this way we are able surely to relieve the congested condition which is so distressing, and no doubt, by diminishing the sensitiveness of the peripheral nerve filaments here distributed, to cure the reflex attacks of coughs which have proved so distressing.

It is most important, however, in just such instances to avoid overloading the child's stomach at bed-time with heavy, rich, or, indeed, too abundant food. A light supper, mainly composed of bread and milk, with a little stewed fruit, is about all that such a child should be allowed to take at its evening meal. If the liver be engorged from a too large food supply, the result is temporary blocking of the circulation; and hence, in many cases, nasal obstruction and cough. Am I not borne out in my statement when many of us acknowledge that certain cases of frequent, obstinate nasal hemorrhage are only permanently arrested by a rigid dietary and repeated counter-irritation, or depletion over the hepatic region?

Just in the same way as a hyper-sensitive area may be discovered in some portion of the nasal passages or nasopharyngeal space, so I find occasionally sensitive areas in the pharynx, in the tonsillar region, upon the soft palate, in the hyoid, or epiglottidean fossa, which will occasion cough as soon as we touch the irritable point.

In what manner it is best to destroy these areas of cough is hard to affirm absolutely. Sometimes I have found one agent, sometimes another, relieve most. Nor is it always true that astringent or caustic applications will do better than soothing anodyne ones, or *vice versa*.

All local remedies at times remain futile, and cough persists and annoys until an entire change of air and scene are obtained.

Of the internal remedies from which I have derived most benefit, I

would mention codeia and terpin hydrate. Codeia does not simply relieve hyper-sensitiveness for a while, it is also directly curative; moreover, it does not constipate the bowels much, as a rule, or upset the stomach, as morphine or opium almost invariable do. Terpin hydrate may have, in addition to its well-known modifying action on diseased mucous membrane, a mild antimicrobic power that perhaps is useful. It always remains true that codeia in doses of gr. $\frac{1}{16}$, more or less frequently repeated, and terpin hydrate in tablet form of 1, or 2 grs. each, every two or three hours, given internally, have been of great service in my hands.

I have not been able to determine invariably the cause of these sensative areas. I meet them occasionally in young girls of marked nervous temperament, who are also anæmic and somewhat exhausted from too much work, study, or pleasure. I also encounter them when the general health is excellent, and it is impossible to get at a satisfactory efficient cause.

Every practitioner is familiar, at least theoretically, with the fact that paroxysmal cough may be occasioned by irritation in the auditory canal. Most physicians have known the mere introduction of a probe or ear speculum to be followed by an outbreak of cough, which only terminated when the offending instrument was withdrawn. Sometimes the condition of the ear is such that we can readily account for cough produced by examination, or, indeed, for the cough which previous to the aural examination had remained a great mystery. Frequently, an impacted mass of cerumen explains the cough, apparently, and after complete removal of this substance the cough will speedily disappear.

There are numerous occasions, however, in which there are no impacted cerumen and no symptoms of aural disease prior to direct investigation by the physician. Then it is, and only then, that we first discover that there is some impairment of the auditory function. But what interests us particularly to state is that, one or more points of the auditory canal are especially sensitive, and appear to have some connection with the appearance or continuance of the cough. In any event, when the sensitiveness of the aural canal is diminished by suitable local applications, the cough tends to diminish or disappear. The point most frequently sensitive is that on the posterior inferior wall of the canal very close to the membrane of the tympanum. Accompanying this sensitiveness, there is pronounced redness of the surface of the canal, with slight furfuraceous exfoliation of the cutaneous layer, which shows distinctly inflammatory action.

Repeated applications of alcohol, or a mild solution of bichloride of mercury (1–1000), or of nitrate of silver (1–100), will cure this condition after a time, as well as ameliorate, if not cure, the paroxysmal cough from which the patient suffers. In many such cases, however, there is a

marked lithæmic condition, and we will help not only the condition of the auditory canal, but also the secondary or concomitant congestion of the pharynx and larynx by frequent doses internally of lithia and bicarbonate of potash, together with some heart tonic like caffeine, which is also useful in promoting urinary excretion.

Of course, in many cases like those of which I have been writing, the aural inflammation and a catarrhal condition of the upper air-passages, with marked increase of secretion, may exist together, and it is almost impossible to say that the ear is in any sense the source of the cough, as this symptom may be wholly caused by an independent laryngitis or tracheitis which is present.

In speaking on this subject of aural reflexes, it may be remarked with a feeling akin to surprise how no attention is paid to it in late editions of works on aural disease, like Politzer and Buck; Dench, it is true, speaks about the reflexes originating in the auditory canal, in his chapter on impacted cerumen, but, so far as I could discover, nowhere else;[1] and yet it is a matter of common knowledge, almost, that "ear cough" exists. Despite this statement, I trust in a practical way my reference to it in this paper may still be found suggestive and useful.

One of the most interesting facts connected with cough—originating evidently in the upper air-passages—is how little we can judge, at times, of the source or nature of the cough from visible appearances; some of the worst-looking throats give, at times, literally no symptoms, and, for one, I am disposed to regard these throats as usually, if not always, normal. Symptoms are evidences of disordered function, and are appreciated by our organs of sense and the reports given us by the patients themselves. If, therefore, none can be found and none are accused, is not this sufficient proof that the organ itself is probably healthy? Again, we shall have all the appearances of a healthy mucous membrane, and yet, strange to say, the patient is always complaining of abnormal or unpleasant sensations, or functional disability in the vocal muscles. These statements are not always exaggerated; they are probably often true, and, if rightly interpreted, lead us to a correct explanation and treatment of them.

Many years ago I reported a case of chronic laryngitis, which served to illustrate how the mucous membrane of the vocal cords may be in an objectively morbid condition, though their physiological functions be restored (AMERICAN JOURNAL OF THE MEDICAL SCIENCES, October, 1875). In an analogous way, I might also show at present how certain patients affected with redness and swelling of the larynx cough a great deal, and others do not. Of course, we can readily affirm that in the

[1] I do not wish to mislead, so that I would add that Dench has a most excellent chapter on "Aural Reflexes," but all due to disease elsewhere, and not in the canal itself.

one case there is some point of continuous irritation elsewhere which causes constant cough, and in the other there is not. Such explanation is, however, nothing more at times than a surmise, and we are thrown back upon our inadequate knowledge at present to give a complete and satisfactory solution of such examples.

In just such instances I have found the best curative effects in a change of air and scene. I am not confident that it is of essential importance that the change shall be from the city to the country, or to a seaside resort. Sometimes the change from one city to another will rid the patient of an obstinate cough which may have lasted for weeks, and resisted much and various medication. Frequently, however, I send these patients inland to breathe the air of pine forests, and where the soil is porous and well drained, and thus obtain most satisfactory results. I know, however, of one seashore resort, *i. e.*, Atlantic City, N. J., which even during the winter months has been most beneficial to cases of "cough" which had resisted other remedial agencies. The special value of this shore climate appears to consist, singular to relate, in its relative dryness as compared with many other places on the ocean.

In the early spring there is nothing which will remove a harassing cough of this kind sooner than a few days' trout-fishing with rod and reel. In the summer, when I am able, I like to send those who cough obstinately to some good sulphur-spring, as I am satisfied that not only the air in the vicinity of sulphur-springs, but also the sulphur-baths and inhalations are very useful in building up impaired nervous constitutions in which such coughs often predominate.

Alongside of these individuals there are others who are apparently in good health and yet are constantly hawking, coughing, and expectorating. Usually these patients are lithæmic to an intense degree, and after a while the lithæmic state is complicated by the presence of an elongated palate and a thickened, congested pharynx and larynx. Lithæmia, if continuously neglected, may become, or find expression in, an evident rheumatic or gouty state. Under these circumstances it is not uncommon to find the mucous membrane of the upper air-passages much infiltrated. Frequently this extends far down the trachea, and tenacious mucus is pretty constantly present and is expelled with difficulty.

Such a condition and the cough dependent upon it is helped more by salicylate of soda or the salts of lithia than by local applications or anodyne cough mixtures. I have already on a former occasion pointed out the marked influence of malaria in producing congestive conditions of the respiratory passages which occasion rebellious cough. This manifestation is often with difficulty traced to its cause, and medication alone seems to establish the diagnosis.

In a few such cases it has seemed evident to me that quinine and

arsenic would not benefit, when bark, in tincture or fluid extract, frequently repeated and in sufficient doses, was certainly curative.[1]

The obstinate cough due to a dilated heart, or one affected at the orifices with organic changes, is very frequent and should be constantly kept in mind. Not seldom, when I have not known what else to do, I have freely stimulated a somewhat weak cardiac action and thus stopped a bad cough in a few days. Previously the patient had taken numerous medicines, with little or no benefit. It is not always possible to make out any cardiac murmur, but it is indicated in these cases occasionally to give heart tonics, even more than if a loud murmur were present. The result is indeed very surprising at times, and the cough disappears very rapidly.[2]

Instead of the rheumatic dyscrasia affecting the joints it may lodge itself in serous membranes like the pleura or peritoneum. In attacking the pleura it produces only slight pain at times, and for this reason, doubtless, no recognition of the cause of cough is made out. I have also known a case where the ear could detect little or nothing by auscultation, and yet I felt sure, through repeated observations and treatment, that pleuritis was the cause of the cough. One or two small fly-blisters locally applied in the beginning aborted the attacks and very soon the patient was well. Sometimes the merely mechanical action of an enlarged spleen or liver pressing on the diaphragm will occasion cough. By slightly forcing these organs downward and inward, a paroxysm of cough may also be occasionally produced. In these instances, a specially sensitive area is found over the lower margin of the liver or spleen.

In many instances of cough of various kinds I have obtained very great temporary relief from dry vapor inhalations of different volatile fluids. The best combination of this kind which I have hitherto discovered, consists of equal parts of camphor, menthol, and eucalyptus. This I use as an inhalation both for throat and nose. The addition of spirits of chloroform, as we all know, to these inhalations will be found often very useful.

In all cases of cough arising from severe bronchial inflammation, or in those from broncho-pneumonia, I am now strongly in favor of using inhalations of beechwood creosote mixed with steam. They are valuable in the cure of these diseases and relieve cough very much.

No doubt the antimicrobic action of the creosote is serviceable. In several instances of grip that I have treated I am thoroughly convinced

[1] In this connection I would remark that certain cases of pneumonia are evidently malarial in origin. In one case I can now distinctly remember, in which Dr. Loomis was the consultant, the typical signs of pneumonia disappeared within twenty-four hours under antimalarial treatment.

[2] I meet occasionally with cases of evidently slight cardiac dilatation, characterized by a systolic bruit, which appear and disappear in a brief period. Fatigue causes them; rest and cardiac tonics cure them, at least for a time.

that the cough of this disease was diminished more with inhalations of creosote than in any other manner. As a preventive means of treatment of cough I have no doubt in my mind that a resort like Hot Springs, Virginia, where we now are, is most useful. By making the skin and kidneys more active, and stimulating the function of the liver, these baths and the massage treatment which follows must render great service. In the bracing mountain air and the dietary control exercised by the resident physician we have additional useful influences.

SYPHILIS AND LARDACEOUS DISEASE.

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When discussing the part played by syphilis in the etiology of aneurisms and atheroma of the aorta,[1] I was led to consider a doubtful point in the pathology of tertiary syphilis itself and its relation to lardaceous disease. I cannot help thinking that the relation between syphilis and lardaceous disease is one which throws some light on the pathological process in both affections.

Probably most medical men believe that tertiary manifestations of syphilis are due to the local presence of microbes, although these microbes of syphilis remain still to be discovered.[2] From this point of view gummatous formation in syphilis must be supposed to bear much the same relation to the microbe of syphilis that "caseous pneumonia" and the synovial swelling in tuberculous joint affections do to the bacillus tuberculosis. When the discovery of the real microbe of syphilis takes place, as it must some time, the microbe will almost certainly be found present in tertiary, as well as in primary and secondary manifestations of syphilis, though in the tertiary period it will probably be found almost completely localized to the lesions which it produces.[3] On the other hand, in the supposed sequelæ of syphilis, such as tabes dorsalis, the microbe will probably not be found present, and a clear demarca-

[1] See this Journal,

[2] The supposed discovery of syphilis bacilli by S. Lustgarten (Medizinische Jahrbücher, Vienna, 1885, pp. 89 et seq.) was at first thought to solve the question, but further experiments have failed to confirm Lustgarten's views as to the specific nature of his bacilli. It has been suggested that some of the bacilli found by him in the primary sores may have been "smegma-bacilli," whilst the few found present in internal gummata may really have been tubercle bacilli, a secondary infection having taken place or a tuberculous caseous nodule having been mistaken for a gumma. The whole evidence, as well as that concerning the claims of other microbes, which have been described as possibly specific of syphilis, was carefully discussed by Prof. P. Baumgarten in his Lehrbuch der pathologischen Mykologie, 1890, vol. ii. pp. 681-686.

[3] See also the additional note at the end of the paper.

tion will thus be established between these supposed "post-tertiary" or "quaternary" manifestations of syphilis and the true tertiary lesions.

It might be argued that because syphilitic gummata are apparently not infectious they cannot contain the microbe of syphilis. The fact, however, that the secretions from tertiary syphilitic ulcers have not been shown to be infectious is no proof that the local presence of a microbe is not the cause of tertiary lesions. If it had not been that a special method of staining had rendered it comparatively easy to detect the presence of the tubercle bacillus, and if there had not been animals easily infected by inoculation with the tubercle bacillus, the presence of this bacillus would never have been proved to be the cause of lupus.

It would seem as if the undiscovered microbe of syphilis, after it has run its usual course and worn itself out in the secondary stage, often remains in a latent or quiescent state scattered about the tissues of the body,[1] and that although unable to give rise to general symptoms in its host, it nevertheless retains the power of causing local harm by gummatous formation, especially when the tissues of the body have become weakened either by general causes, such as alcoholism, or by local causes, such as a traumatism limited to some special part of the body.

Many considerations are in favor of the view that gummata are caused by the local action of a microbe. I shall here confine myself to putting forward an argument in favor of this view which has not yet, as far as I know, been brought forward. It must be taken only for what it is worth, but, as fresh evidence, may be of provisional use, until the question be finally settled by the discovery of the real microbe.

Lardaceous disease is acknowledged to occur in syphilitic patients at times without any suppuration, but besides this fact there exists another curious connection between syphilis and lardaceous disease, namely, that a localized lardaceous formation in the neighborhood of visceral gummata may sometimes be recognized;[2] the latter occurrence, although hardly

[1] There can be no doubt that methyl violet is a far more reliable stain than iodine for the lardaceous material; so Cohnheim and Cornil found it, and this is the opinion of N. P. Krawkow, who has recently worked on the etiology of the lardaceous formation (Cent. für Allg. Path. und path. Anat., vol. vi. p. 338). Lesser degrees of lardaceous disease would often escape observation but for careful microscopical examination, and doubtless for want of microscopical examination they often do remain unobserved, especially in the kidneys. I would point out that a routine series of microscopical examinations for early stages of lardaceous disease should be made in the kidneys from cases of chronic parenchymatous nephritis and "mixed" nephritis. This pathological anatomical study is required just as much as are further experiments on the production in animals of lardaceous disease before the laws determining the formation of this pathological material in the tissues of human beings can be finally determined. It is possible that there may be some kinds of chronic parenchymatous nephritis always associated with a slight lardaceous change in the kidneys, and that in such cases the nephritis and the lardaceous change may be both due to the excretion by the kidneys of toxins produced by the action of microbes in other parts of the body, for instance, by pyogenic microbes in the lungs of advanced cases of chronic pulmonary tuberculosis.

[2] See also the additional note at the end of this paper.

noted by authors, is very striking when observed. It is my intention here to use these facts as fresh evidence in favor of the view that a microbe is present in tertiary syphilitic lesions. I will endeavor to make myself clear.

By the recent experiments of Dr. N. P. Krawkow (*Cent. für Allg. Path. und path. Anat.*, May 20, 1895) it has certainly been made clear that lardaceous disease is usually the result of absorption into the blood of products of the metabolism of parasitic microbes. Krawkow failed to produce lardaceous disease in animals when the suppuration was induced by turpentine instead of by pyogenic microbes,[1] whereas he actually succeeded in inducing lardaceous formation in the tissues of a rabbit by repeated injections of the mere filtrate of a culture of the bacillus pyocyaneus, that is, by the metabolic products of a microbe without the microbes themselves.[2]

In human beings lardaceous disease hardly occurs without definite suppuration except in cases of syphilis. Krawkow seems to have shown that when it arises from suppuration it is due to the microbes of suppuration. There seems to be little doubt that it occurs sometimes in cases of syphilis when there has been no suppuration at all or only very slight suppuration; it seems, therefore, not unreasonable to suppose that in such cases of syphilis the lardaceous formation is due likewise to a microbe, although not a pyogenic microbe, namely, to the as yet undiscovered microbe of syphilis.

The occasional localization of the lardaceous change in the neighborhood of syphilitic gummata may be taken as further evidence both that gummata are due to the local presence of the microbe of syphilis (whatever the microbe may ultimately turn out to be), and that this microbe has the power of causing lardaceous disease without the help of pyogenic microbes. This further evidence depends on the fact, as pointed out below, that if these theories be accepted, the localization of lardaceous formation in the neighborhood of syphilitic gummata can easily be explained, whereas it cannot be so easily explained by any other theories.

Granting that a gumma is a formation caused by the local presence of a specific microbe of syphilis (just as a tubercle is caused by the local presence of the tubercle bacillus), then we can understand that the tissue in the neighborhood of a gumma is more likely to be affected by the products of this microbe than are tissues further removed from

[1] He points out, however, that an abscess induced by the injection of turpentine may easily become secondarily infected with pyogenic microbes, and thus lardaceous disease, readily brought about by the action of these microbes, might be incorrectly attributed to aseptic suppuration caused by turpentine.

[2] Krawkow suggests that the lardaceous material may be a product of this microbic metabolism, and follows those who think that it is deposited in the affected tissues by a process of degeneration.

the gumma. The metabolic products of the microbes inhabiting and causing the gumma must to some extent leak out and infiltrate the neighboring tissues, before being carried off by the lymphatics. If, as seems probable, one of the properties of these microbic products is to cause lardaceous formation in certain tissues (especially the walls of the minute bloodvessels) on which they can act, then it is not surprising that the lardaceous change should occasionally be found especially marked in the immediate neighborhood of gummata, or that it should take place there before occurring in more distant parts. In other words, it seems likely that the microbic products, manufactured within the gumma, should affect the surrounding parts before being carried off into the general circulation; after which these microbic products gradually produce the characteristic changes of lardaceous disease in the spleen and other organs peculiarly liable to this change before they are finally excreted by the kidneys.

In this short paper I have endeavored to show how lardaceous formation in the tissues, when occurring in association with active tertiary syphilitic manifestations, and especially the lardaceous formation occurring in the immediate neighborhood of gummata, may be taken as additional evidence of the already probable view that active tertiary syphilitic manifestations are caused by the presence of a specific microbe at the site of the lesion. The conclusions which appear most probable may, I believe, be summed up thus:

That there is a specific microbe of syphilis, the local presence of which is the cause of the tertiary lesions of syphilis; that the metabolic products of this microbe have, like those of some other microbes, such as the staphylococcus pyogenes aureus and the bacillus pyocyaneus, the peculiar property of producing lardaceous formation in the tissues of the hosts.

Additional note on the view that tertiary manifestations of syphilis are caused by the local presence of a specific microbe.

By those who held that syphilis when it has reached the tertiary stage cannot be transmitted by inheritance from parents to their children, it might formerly have been alleged that the exceptions to this rule are apparent only, and that really in such apparent exceptions remnants of secondary syphilis must have existed in one of the parents. This explanation is, however, now untenable, for no one would call syphilis after it has existed fifteen years secondary syphilis, and cases are known where parents fifteen years or more after the commencement of syphilis have transmitted it to their offspring. Professor Fournier proves this by instances, and considers that of cases where syphilis has been trans-

mitted to the fœtus, in about one case out of ten the parental syphilis dates from six years back or more.[1]

If, on the other hand, syphilis be allowed to be due to a microbe which, after the secondary stage, is supposed often to remain in the body in a latent or almost latent condition (though able to become active again as the producer of the local manifestations in tertiary syphilis), then the whole matter may be more easily explained.

According to this view, after the secondary period of syphilis is over, the surviving microbes of syphilis are supposed to be lying quiescent in different tissues of the body, but may occasionally be washed out by the blood-stream into the general circulation. If we accept this explanation it does not seem astonishing that parents while suffering from active tertiary syphilis produce children untainted by syphilis, just as parents whilst suffering from active tuberculous disease produce children perfectly free of congenital tuberculosis. Neither is it surprising that children of parents suffering from active or even latent tertiary syphilis may at times be born with congenital syphilis, in the same way that children of mothers suffering from active tuberculosis may be occasionally born with congenital tuberculosis, as has been conclusively proved by the observations of Birch-Hirschfeld and Schmorl (*Beiträge zur path. Anat.*, 1891, vol. ix. p. 428); P. Londe (*Rev. de la Tuberculose*, 1893); and Schmorl and Kockel (*Beiträge zur path. Anat.*, 1894, vol. xvi. p. 313).

In fact, just as syphilitic gummata and gummatous infiltration may be compared to tuberculous caseous nodules and tuberculous disease of synovial membranes, so, according to this theory, the process by which the fœtus may become infected from a mother in the tertiary period of syphilis may be compared to the process which Schmorl, Birch-Hirschfeld, etc., believe to be the one by which tuberculosis spreads from the mother to the fœtus. Professor Birch-Hirschfeld[2] suggested that in cases of congenital tuberculosis the tubercle bacilli "grew through" from the maternal into the fœtal portion of the placenta, and thus infected the fœtal circulation; this view has, moreover, gained support from the observations of R. Kockel and M. Lungwitz on "Placental Tuberculosis in Cows" (*Beiträge zur path. Anat.*, 1894, vol. xvi. pp. 294 *et seq.*).

We can easily understand, then, that if there be any microbes of

[1] Out of 562 cases where the pernicious effects of hereditary syphilis showed themselves, in sixty cases the parental syphilis was acquired six years previously or more. See Professor Alfred Fournier, L'Hérédité Syphilitique, Paris, 1891, p. 122.

[2] This was from analogy with what was observed to take place in the case of pregnant animals inoculated with anthrax. See Birch-Hirschfeld, "Ueber die Pforten der placentaren Infection des Fœtus," in Beiträge zur path. Anat., 1891, vol. ix. pp. 383 *et seq.* Birch-Hirschfeld suggested that the process of infection of the fœtus in congenital syphilis and congenital tuberculosis might be similar to that observed by him in the case of anthrax.

syphilis in the general circulation of a woman at some period of her pregnancy, these microbes may reach and be lodged in the maternal portion of the placenta; there they may develop and multiply locally and thus "grow through" into the fœtal portion of the placenta, where some may enter the fœtal circulation and give rise to congenital syphilis.

This theory, moreover, explains why a quiescent syphilis in the mother is probably more likely to be transmitted to the offspring than a quiescent syphilis in the father, for in the case of the mother there is the long period of pregnancy, during which the ovum may become infected in the way described. This theory will also elucidate cases, where a healthy child is born of syphilitic parents (not necessarily undergoing anti-syphilitic treatment at the time), though subsequent children suffer from congenital syphilis.[1] In such a case one must suppose that during the earlier pregnancy the syphilis microbes were lying quiescent in the mother's body, or at least that any which chanced to get into the general circulation failed to infect the placenta; but that during subsequent pregnancies the fœtus became infected either by some of the (previously quiescent) microbes getting into the general circulation and lodging in the placenta, or else by some tertiary syphilitic process localized in the neighborhood, reaching the fœtal circulation by spreading through the placenta.

By analogy mercury may be supposed to owe its influence on syphilis to some specific action on the microbe. No one will question the striking use of mercury seen at times in cases of tertiary syphilis as well as in its earlier stages. Those who think that primary and secondary syphilis are due to the presence of a microbe, on which mercury exercises an antagonistic action, must therefore admit that the beneficial effect of mercury in tertiary syphilis may be used as a slight argument in favor of the view that the tertiary as well as the primary and secondary manifestations of syphilis are due to the presence of a specific microbe. It would, however, be unprofitable here to discuss at greater length well-known evidence in support of a view which already appears probable; so probable, in fact, that the only additional evidence any longer really needed is the evidence which would convert the theory into a fact, namely, the discovery of the microbe itself.

[1] See Fournier, op. cit. pp. 263-267.

IODOFORM-OINTMENT INJECTIONS IN THE TREATMENT OF SUPPURATIVE ADENITIS OF THE GROIN.[1]

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The chief objects to be obtained in the treatment of suppurating inguinal buboes are their rapid disappearance, an amelioration of pain, and the absence of a compromising scar in the groin.

To this end various surgical procedures have been suggested from time to time, but it was not until 1856, when Broca,[2] Roux, Marchal, Abeille, and others advocated aspiration of the pus and injection of diluted tincture of iodine into the abscess cavity, that such favorable results were in a degree obtained.

In 1857 Lebert[3] reported a number of successful cases treated in a similar manner, except that he evacuated the pus through a small incision and injected the pure tincture of iodine.

Again, in 1871, Teackle[4] speaks of this method as being remarkably successful, and quotes several cases in which he has employed it.

Scott Helm,[5] in 1886, was, I believe, the first to substitute iodoform for tincture of iodine, and used it in the following manner:

The parts were cleansed and anæsthetized by means of ether spray, the pus was drawn off with a hypodermic, and the abscess cavity washed out with a weak solution of carbolic acid. Iodoform suspended in glycerin was then injected, the needle withdrawn and the puncture closed with collodion; over this an ordinary dressing and spica bandage were applied. Twenty-two cases were treated in this manner with but one failure.

Later, Helm substituted oleic acid for glycerin, and finally iodol for iodoform.

In 1889, Von Eichstoff[6] reported a series of cases treated by injections of iodoform and ether which he employed in two different ways, as follows:

First method. The pus was withdrawn and the cavity irrigated with boric acid solution, and then injected with the iodoform and ether mixture, the puncture being covered with an ordinary dressing, which was

[1] Read before the American Association of Genito-Urinary Surgeons, Niagara Falls, May 28, 1895.

[2] Bull. Gen. de Therapeutique, 1856, li. 208. [3] Wiener med. Wochenschrift, 1858, No. 43.

[4] Medical Record, 1871, vi. 344.

[5] Chicago Medical Journal and Examiner, 1886, liii. No. 3.

[6] Thèse de Paris, Trait. des Bubons, 1889.

left in place for three days. In some cases he had to make a second injection, but never a third. A cure was accomplished in from three to eight days.

Second method. The parts were cleansed and the iodoform-ether mixture injected directly into the abscess cavity without evacuating the pus. As a rule, two injections had to be employed before a cure was accomplished, which on an average took about twelve days.

In the same year Fontan[1] described very minutely the treatment of suppurating buboes by injections of iodoform vaseline. He gives a total of forty-one cases so treated, the average time for cure being six to seven days. His method of procedure is as follows: The parts are shaved and cleaned; the bubo opened with a lancet and all the pus forced out; the abscess sac is then irrigated with diluted Van Swieten's fluid, and iodoform-vaseline injected with a glass syringe, previously warmed in hot water. A cold wet dressing is applied in order to congeal the ointment at the opening. In some cases it was necessary to resort to an injection a second time.

In 1893, W. K. Otis[2] reported very favorable results in sixteen cases in which he used injections of iodoform ointment in a similar manner.

Having employed this plan of treatment with such satisfactory results, I feel justified in reporting the few cases so operated on; these were not selected, but taken at random as they entered the hospital and clinic. The steps in the procedure are similar to those advocated by Fontan, with the addition of the peroxide of hydrogen, and are as follows:

1. The operative field is shaved and rendered surgically clean in the usual manner.

2. A few drops of a 4 per cent. solution of cocaine are injected beneath the skin where the puncture is to be made.

3. A straight sharp-pointed bistoury is then thrust well into the most prominent part of the tumor until pus flows.

4. All of the pus is forced out through this opening by firm but gentle pressure, as this procedure is, as a rule, very painful.

5. The abscess cavity is irrigated with pure peroxide of hydrogen until it returns particularly clear.

6. It is then irrigated with 1 : 5000 bichloride of mercury solution, all of which is carefully squeezed out.

7. The now thoroughly cleansed abscess cavity is completely filled, but not painfully distended, with 10 per cent. iodoform ointment by means of an ordinary conical glass syringe, previously warmed in hot water.

8. A cold wet bichloride dressing is applied with a fairly firm spica

[1] Espieux: Thèse de Montpellier, April, 1889. Archives de Méd. Navale, 1889, lii. 5.
[2] Journ. Cut. and Genito-Urin. Dis., May, 1893.

bandage, the cold congealing the ointment at the wound, and thus preventing its escape into the dressing.

The patient should be kept very quiet for the first twenty-four to forty-eight hours, rest in bed being preferable, although not absolutely necessary.

The dressing is removed at the end of the third or fourth day and the parts examined; if pus has reaccumulated or the ointment escaped into the dressing, a second injection may be made. If, on the other hand, all looks well, the first dressing is replaced by a gauze pad and spica bandage, and the patient told to report in two or three days for examination.

In a few of the above series of cases several drops of an oily serous fluid were observed at the first dressing escaping from the puncture; this fluid was carefully wiped off and a sterilized gauze dressing applied, recovery being in no way retarded by the exudation.

The following table gives the etiology, amount of pus evacuated, number of injections employed, and time for cure in each case:

Case.	Etiology.	Amount of pus.	Number of injections.	Result.
1	Gonorrhœa: chancroids . .	12 drachms.	One.	Cure in 14 days.
2	Gonorrhœa	4 "	One.	" 10 "
3	Chancroid	12 "	One.	" 14 "
4	Chancroids	3 "	Two.	" 7 "
5	Gonorrhœa	6 "	One.	" 14 "
6	Chancre: chancroids . .	11 "	One.	" 7 "
7	Chancroid	4 ounces.	One.	" 21 "
8	Chancre: chancroid . . .	2 "	Two.	" 21 "
9	Chancroids	12 drachms.	One.	" 7 "
10	Chancroid	6 "	One.	" 14 "
11	Gonorrhœa	6 "	One.	" 8 "
12	Gonorrhœa	4 "	One.	" 10 "
13	Chancroid	3 "	One.	" 11 "
14	Gonorrhœa: chancroid . .	5 "	One.	" 12 "
15	Gonorrhœa	12 "	One.	" 12 "

Suppurative action and pain ceased after one injection of the iodoform ointment, except in two cases; in these the injection had to be repeated on account of a slight reaccumulation of pus.

None of the above cases were pronounced cured until firm pressure in the groin elicited neither pain nor fluctuation, and the overlying skin had resumed its normal appearance.

In order to secure the most favorable results from this method, it should only be employed when the glands are quite thoroughly broken down, so that the iodoform may come in direct contact with all of the infected tissues.

It is not, of course, claimed that this method will succeed in every case of suppurative adenitis, but its many advantages over the more

radical procedure render it, in my judgment, worthy of a trial. There is no necessity for the patient to remain in bed, or undergo anæsthesia; the little scar in the groin is soon covered by hair, and he or she is spared a more or less painful and tedious convalescence. If after one, two, or even three injections, this method fails to produce the desired result, an incision may then be made and the contents of the bubo removed; the previous treatment not having interfered in any way with this operation.

FEVER IN THE COURSE OF BRIGHT'S DISEASE AND IN URÆMIA.

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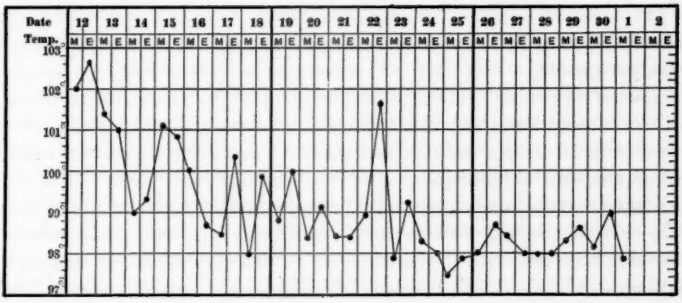
Among the many and various symptoms of acute and chronic nephritis that have received special attention the temperature has, comparatively speaking, been neglected, though a few authors have given it considerable study. These will be alluded to more particularly in discussing the various conditions under which fever may occur and the forms it is apt to assume.

There are three groups of cases of distinct character in which acute or chronic nephritis and elevation of the body temperature are found to be associated. In the first group are included cases of mild febrile reaction at the beginning of acute nephritis; in the second the elevated temperatures result from complicating diseases, while the third and least well recognized group is that in which the temperature is elevated in the course of the uræmic state. It is to the latter that I wish to call attention more particularly in this paper, but it will be interesting, I think, to consider briefly the other groups for the sake of completeness.

I. The temperature at the onset of acute Bright's disease has quite commonly been found to be elevated. This elevation is rarely marked enough to attract more than passing attention, and little study has been made of its causes. Without attempting to enter upon the broad question of the pathogenesis of Bright's disease, we may say that this early slight fever is due either to the inflammatory or degenerative lesion of the kidney, to poisoning of the system, on account of the retention of deleterious substances which the kidney, as a rule, excretes, or, as would sometimes seem probable, from the primary infection or intoxication, which is to be looked upon rather as the cause than as the result of the disease of the kidney. The clinical fact, however, is well known. In the cases appended the type and degree of pyrexia usually observed are well illustrated.

CASE I.—W. C., aged thirty-five years, had been out of bed but a few days after recovery from an attack of typhoid fever when he became languid, weak, and, as he expressed his condition, very stupid. In this state he first came under my observation. The urine was greatly reduced in quantity and contained considerable albumin with casts. The man's temperature was at first 102.6° F. It soon declined, however, but remained moderately elevated for over a week, subsiding gradually. During this time there was also a gradual increase in the quantity of the urine, and the patient's general condition steadily improved. He recovered entirely in a short time.

CHART I.



CASE II.—M. D., aged thirty-five years, had noticed for several months increasing difficulty in putting on his shoes, but he really had not thought himself ill until three weeks before his entrance into the hospital. At that time there was decided swelling of the feet, which later involved the legs and scrotum, and was soon followed by vomiting, pain inthe back, and headache. The urine was scanty, acid in reaction, and contained considerable albumin. The specific gravity was 1020. The patient was extremely pale, and had decidedly the appearance of a nephritic. The arteries were a little stiffened and the ventricle hypertrophied. After a few weeks the dropsy and other symptoms had improved so much that the patient was discharged from the hospital. In this case it seemed evident that the man had a chronic form of nephritis, and that the present attack was of the nature of an acute congestive or inflammatory exacerbation. There were none of the ordinary indications of a uræmic state. The temperature in this case, also, was above the normal for several days, but fell as the urine increased in quantity and the dropsy disappeared.

CASE III.—M. C., a young woman, having a subacute form of nephritis was exposed to the weather and became thoroughly chilled. Shortly afterward there was considerable pain in the back, and the urine showed increase of albumin, with blood cells. The temperature at the same time rose to 102° F., and varied between this and the normal for two days.

Such degrees of fever at the onset of acute nephritis are usual and well known. In some instances, however, the primary fever is very

marked, and may be quite alarming. This is especially the case in the primary bacterial nephritis of Bamberger, Aufrecht, Litten, Babes, Perret, and Mannaberg, or infectious nephritis, as Friessinger calls it. In these cases, indeed, there is little doubt but that these are instances of infectious diseases localizing themselves in the kidneys from the first. Sometimes, as in the series reported by Friessinger, there is distinct epidemicity, and various micro-organisms have been isolated in such cases. It is in these infectious cases that high fever may occur at the onset. Friessinger[1] records one in which it reached 105° F., and in which the symptoms of infection were very marked.

II. A second variety of fever in the course of nephritis is that which owes its origin to inflammatory complications, such as pericarditis, pleuritis, pneumonia, or the like. There may also be temporary elevations of the temperature, resulting from some renal complication, such as acute exacerbations in chronic nephritis, the development of pyelitis, or the deposit of chalky material in the course of a gouty Bright's disease. Sometimes these inflammatory complications may be so latent that their existence remains unrecognized, and the cause of the elevated temperature is undiscovered until autopsy is performed. Some authors have invoked this tendency to latency of inflammatory complications to explain the high fever of some of the forms of uræmia presently to be described; but it is to be noted that the fever resulting from inflammatory complications in Bright's disease are apt to be very low, especially in comparison with that seen in similar conditions in the course of rheumatism, whereas the cases to which this theory has been applied are just the ones in which hyperpyrexia is most commonly observed. I do not dwell longer on this question, as it is, after all, aside from the present discussion, and for the same reason it is unnecessary to instance cases.

III. The third condition under which fever is likely to occur in the course of Bright's disease, and it is to this I have wished most particularly to call attention, is the state ordinarily known as uræmia. Before entering upon this question in its clinical aspects it may be of interest to review hastily the various theories that have been offered in explanation of this condition. One of the oldest and best known is that of Traube. This would explain the manifestations of uræmia on a mechanical basis, ascribing them to a state of repletion of the vessels and œdema of the brain, inducing a secondary condition of anæmia of the brain substance. This theory was based on the discovery in some cases of conditions such as described, particularly in autopsies on persons dead of uræmic convulsions; and it seemed to receive a measure of support from the investigations of Munck, and Otto and Bidder, who found that injection of water into the cervical vessels induced symptoms not unlike those of uræmia. The theory, however, is disproved by the fact that many cases

do not present the œdematous conditions of the membranes or brain-substance described, and more particularly by the observation of Carter,[2] of Liverpool, that analysis of the cerebral substance does not show a disproportionate amount of serosity after uræmic convulsions. The experimental evidence cited loses its force when we recognize that the quantities of water injected are alone capable of inducing toxic manifestations. After Traube various chemical theories have been brought forward. One thought that ammonia was the noxious agent, another ascribed the symptoms to potassium poisoning, another to creatin, creatinin, urea, and so on. That none of these theories is satisfactory to explain the manifestations of uræmia is apparent enough when we study the experiments of the many who have worked in this field. It is true that each of these substances may act as a toxic agent when injected in sufficient quantity, but none is capable of reproducing the exact manifestations of uræmia. Bouchard,[3] who has done so much in this field, thinks that there are at least seven toxic substances in the normal urine. These are: 1, a diuretic substance (urea); 2, a narcotic poison; 3, a sialogogue; 4, a temperature-reducing substance; 5 and 6, convulsants (one, doubtless, potassium), and 7, a poison which causes contraction of the pupil. However this may be, and no doubt all of these substances do exist in the urine, none of them can be looked upon as the cause of uræmia. Recently Hughes and Carter[4] have added very important facts to the elucidation of the nature of uræmia. According to them the active poison is probably an albuminous substance quite different from any which exists in the normal urine.

In all of these investigations it has been assumed that the temperature in uræmia is reduced, and no doubt this is usually the case. A few investigators have, however, studied the question of urine-intoxication with special reference to the temperature. Binet[5] showed that human urine contains a substance which may be precipitated, dissolved in glycerin and reprecipitated with alcohol, and which has the power of causing elevation of the temperature in guinea-pigs. It is most abundant in the urine of tuberculous individuals, and is most active in tuberculous guinea-pigs. The temperature usually begins to rise after two hours, reaches its height in about three hours, and subsides after four or five. He regards it as of the nature of a ferment, and calls attention to the fact that various ferments (pepsin and trypsin, Bergmann and Angerer, fibrin ferment, Edelberg, bacterial ferments, Charrin, Ruffer, Hildebrand, Roussy and others) have been found to have similar pyretogenous properties, and that some of these occur in the urine at times. Lepine[6] sought to escape some of the objections that may be urged against other methods by injecting sterile normal salt solution into the ureters of a dog under pressure sufficient to insure a steady entrance of the liquid. Injection of the same amount of sterile salt solution directly into the veins of a dog did

not cause the symptoms (dyspnœa and fever), and he concludes that there are pyretogenous substances in the urine. He was further able to induce fever by injection of extract of the kidneys of a normal dog. This experiment is open to several objections. Bouchard, and latterly Hughes and Carter, injected the urine itself into the veins, and found very constantly fall of the temperature. Binet would explain this by the assumption that there are both fever-provoking and temperature-reducing substances in the urine, and that the latter usually dominate.

Clinically, the occurrence of fever in the course of uræmia has been known to exist for a long time, but little attention was paid to it. Lees called attention to it in 1852, but authors generally rarely referred to the temperature until the publication of the well-known papers of Bourneville,[7] in 1872. This author claimed that depression of the body-heat is a constant phenomenon of uræmia, and, indeed, made this the basis for denying the uræmic nature of eclampsia. Later writers, all or most, at least, agree that many cases of uræmia are attended with elevation of temperature. This change of view is chiefly attributable to the writings of Bartels,[8] who maintained that he had seen but one case of uræmic convulsions in which the temperature was not elevated. There is, however, much difference of opinion as to the cause of the fever, many holding that it is usually due to complications. This view, while it is true of some cases, is not true of most, for numerous instances are recorded where careful search was made after death for some local lesion to explain the fever. I think that the majority of authorities would now admit without question the existence of fever of purely uræmic origin, and the tendency of the day is to assign as the cause of this some toxic excitation of the heat-regulating apparatus, rather than cerebral or meningeal congestion, or the like. One very striking evidence of the toxic, rather than organic, character of the fever in uræmia is the precise manner in which the temperature frequently follows the increase or decrease of the uræmic state and the very rapidity of the rise and decline. That congestion of the meninges or œdema of the brain is not the immediate cause of the fever is evident from the facts that, while in cases reported by Chantemesse and Tenneson,[9] and by Richardiere and Therese,[10] there was one or the other of these conditions; in those of Bouveret[11] and of Dumont[12] no lesion was discovered, and Carter, in his chemical analysis, failed to find excess of water in the brain substance.

Bartels called attention to the clinical fact that very frequently uræmic symptoms develop rapidly when there is rapid subsidence of the dropsy, and ascribes this to the liberation of poisonous substances contained in the cellular œdema. In such cases there is apt to be some elevation of temperature, which Bartels would ascribe to the intoxication; but Carter thinks this due rather to the hot vapor baths, and cites

a case in point. That this view is not an accurate one is apparent in the following case under my observation. In this there was decided rise of temperature coincidently with the rapid decline of dropsy, and for which there was no adequate explanation excepting that indicated. No hot-air bath had been used, and the temperature became normal as soon as the œdema was completely discharged.

Case IV.—P. M., aged twenty-six years, a driver, had led a very exposed life; had had a chancre two years previous to his coming under my observation; but was not excessive in the use of alcohol, and had really had no serious illness except pneumonia a few years before. Six months before he had pain in the back and other vague symptoms, which disturbed him for a time, but subsided spontaneously. The illness which brought him under my notice began on the night of November 6th, after he had been exposed to the weather. He thought he had taken "cold," and had considerable cough. The next day his face was swollen, and later the legs and abdomen. The urine was very scanty, high colored, the specific gravity was 1034, there was much albumin and hyaline, granular and epithelial casts. The anasarca became marked; there was occasionally great dyspnœa. No lesion of the heart or lungs was discovered on physical examination. The treatment instituted was purgation and sweating with pilocarpin. After a day or two of normal temperature there was a slight rise, which soon subsided, and then the temperature was normal for ten days. At the end of that time there was a sudden rise to 101° F., coincident with the establishment for the first time of free diaphoresis by the aid of pilocarpin. The amount of liquid discharged through the skin was truly remarkable, as was also the rapidity with which the anasarca disappeared. During the four or five days in which the dropsy was subsiding the temperature remained elevated, and after the complete disappearance of œdema the temperature became normal, and convalescence was soon complete. The patient was discharged entirely well, and was still so a year later.

Whatever solution may eventually be found for the existence of fever in certain cases of uræmia, I am for the present more concerned with establishing the clinical fact that there are certain cases of uræmia in which the temperature is elevated in the absence of any complication and irrespective of the inflammatory condition of the kidney itself. It is a well-recognized clinical fact, first clearly established by Bourneville, that the temperature in uræmia is usually depressed below the normal, but it should be recognized that the reverse may be the case, and that in the course of chronic Bright's disease a sudden elevation of the temperature without any discoverable cause may be due to uræmia. It is impossible at the present time to say why there is elevation of the temperature in some and depression in others; nor is it possible to indicate any features in which the febrile cases differ from the afebrile excepting the temperature, for exactly the same clinical manifestations that are seen in the febrile cases are often present without any elevation of the temperature. It is possible, however, to distinguish two classes of cases

as far as the temperature is concerned; those in which the onset is very sudden and the symptoms pronounced, and those in which the approach is insidious or the symptoms of a depressive character.

I. The first or eclamptic form is most frequently present in cases of uræmia with convulsions, as Bartels pointed out, but there may be just as decided fever in comatose or delirious cases in which no convulsion occurs. Many cases of this form of uræmic fever are recorded, but I select the following, in which the temperature was specially observed, to indicate the character of the attacks.

Richardiere and Therese report a case of acute nephritis following exposure to cold, in which the temperature from the first was entirely normal, and remained so until the patient developed signs of urœmia. The temperature then rose to 102.2° F., after which convulsions developed and there was a further rise of the temperature to 104° F. After five days there was a critical decline of fever, and the temperature again remained normal until a second attack of uræmia was announced by dimness of vision, headache, and a rise of temperature to 101.2° F. This attack was aborted by prompt venesection. In their second case there were the signs of interstitial nephritis, with œdema of the legs and scrotum. For some time the temperature was normal, then the patient became delirious; he refused to stay in bed, and shortly afterward or coincidentally the temperature suddenly rose to 103.6° F. There was also epistaxis. The day following epileptiform convulsions were noted, and the uræmic state continued for five days, finally yielding to venesection.

Mousson[13] reports a case in which there were dropsy, pleural effusion, considerable albuminuria with blood cells. After five days of normal temperature there was a sudden elevation to 103.6° F. with coma, epistaxis, and convulsions. Subsequently there was Cheyne-Stokes breathing, and the patient was bled. The temperature fell quickly and remained but little above the normal for a day or two, when it subsided completely. In this case the first elevation of the temperature occurred before the first convulsion.

Gillet[14] reports a case in a man of twenty-eight years, in which there was stupor followed by convulsions. The temperature was between 99° and 100° F. There was bloody expectoration and some soft râles at the bases of the lungs; the urine was highly albuminous. The temperature rose during the two days following until it reached 105.8° F., when the patient died. The autopsy disclosed interstitial nephritis, œdema of the bases of the lungs, and some meningeal congestion.

In these cases of typical eclamptic fever it will be noted that the temperature in reality began to rise before the onset of the convulsions, which cannot, therefore, be regarded as causing the elevation of temperature by the violence of the muscular movements. In the first case of

Richardiere and Therese this was further disproved by the subsequent abortive attack in which the temperature rose without convulsions, subsiding promptly after venesection. Similar cases have been recorded by Dumont, Carter, Amidon, Wiener, Hughes, and others. The two following from my own experience are instructive and to the point.

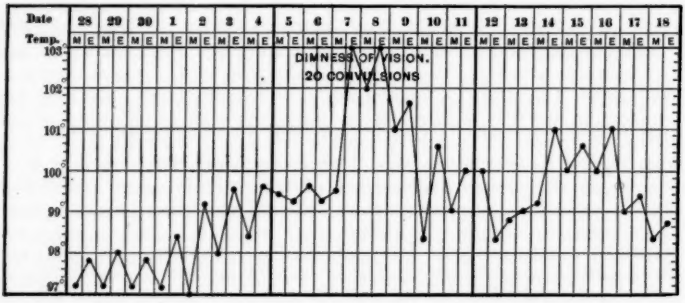
Case V.—T. D., aged thirty-seven years, was brought to the hospita; by the ambulance at 11 A.M. June 6th. He was decidedly stuperousl the breathing was heavy, somewhat stertorous; the skin was moist, the pupils of ordinary size and responsive. The patient was catheterized, and the urine found to be scanty, heavy, and albuminous. The man was evidently in an uræmic condition, and was therefore placed in a vapor bath, and perspired very freely; but despite this favorable action he rapidly sank into a condition of complete unconsciousness and became convulsed. Convulsions followed in rapid succession during two hours after his admission, in spite of the administration of nitrite of amyl, chloroform, and chloral; and the patient was finally bled after the eighth convulsion, nine fluidounces of blood being removed. At once there was decided improvement. An abortive convulsion, which was readily controlled by inhalation of chloroform, occurred five minutes after the venesection, but the patient almost immediately afterward became conscious, and remained so during most of the day. The next night there was another series of convulsions, beginning about ten o'clock. After the fourth the patient was again bled (nine fluidounces), after which there were but two light fits. On the night of the 10th, three days later, there were three slight convulsions, after which the patient recovered. Repeated examinations of the urine confirmed the diagnosis first made of chronic parenchymatous nephritis; and though most careful physical examination was made no complication of any kind was discovered in the other organs. The point of particular interest in this case for the present was the behavior of the temperature. On admission this was found to be 100.1° F., but almost immediately it began to rise. The elevation really began before the first convulsion, but the greatest elevation was not reached until some time after the beginning of the fits, when it was 106.6° F. After the cessation of the convulsions there was a rapid decline of the temperature to about 100° F. The next day there was another rise of the temperature without fits to 102° F., and during the night when the second series of convulsions occurred the temperature rose to 102.6° F. After this the temperature was subnormal until the last series of fits, when it rose again, but only to the normal.

Case VI.—J. O., aged twenty-six years, a laboring man, giving a good family history; had himself led a very irregular life. He had worked in various parts of coal-mines from an early age; was habitually much exposed, and drank excessively of ale. In December of 1892 he became thoroughly wet and chilled and took "cold." This was not unusual, as he had had chronic cough, beginning at a time when he worked in the dust of the "breakers." About January 1, 1893, he put himself under the care of a medical man, but did not improve, and on the contrary noticed that his weight was increasing and that his face was growing puffed. Soon his feet became swollen, and subsequently the whole body, his weight increasing twenty pounds.

When he first came under my care, in February, his appearance was

that of a man having the anasarca of renal disease; his color was very white, the blood examination showing 3,350,000 erythrocytes, 10,000 leukocytes, and 53 per cent. of hæmoglobin; there was a puffed condition of the face and neck and universal œdema. The urine was below the normal in quantity, varying from twenty to forty ounces; it was loaded with albumin and contained hyaline, granular, and blood casts. The patient was troubled with considerable cough, and on examination of his chest there were dry and moist bronchial râles, with evidences of slight emphysema. The heart was healthy, but the second sound markedly accented, and the pulse so tense that it was difficult to determine the existence or absence of hardening of the vessel wall. He was ordered diuretic and purgative treatment, but the condition remained practically unchanged during several months. His temperature during

CHART II.


Date
28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
Temp.
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
103°
102°
101°
100°
99°
98°
97°
DIMNESS OF VISION.
20 CONVULSIONS

this time was almost constantly below the normal, and never above that point. On the first day of May the temperature rose to the normal, and the next day was 99.2 F. At the same time the patient complained of pain in one of his teeth, and then of headache. These symptoms continued for five days, the temperature remaining at about 99° F. On the 7th, in the morning, there was sudden dimness of vision, followed by an uræmic convulsion, and after this by a number of others during the day and the following night—twenty in all. He was put in a hot vapor bath, was bled about twenty fluidounces, was given croton oil, and purged with salines, and received chloral by the rectum. The next day he was wholly conscious and continued to improve. The temperature, which had been about 99° F. before the first convulsion, rose shortly after the onset of the latter to 103° F., where it remained the next day. For several days after this it was elevated, though not so much. Subsequently there was a rise of temperature due to mastoiditis, but excepting this there were no further unpleasant symptoms, the temperature remained normal or below the normal, and the patient finally recovered almost entirely.

In each of these, again, it is noted that the rise of temperature began before the first convulsion and the second one is particularly instructive and significant in that there was a period of five days of slight fever

with mild uraemic symptoms before the convulsions began. Indeed, my previous experiences with febrile temperature before violent uræmia led me to predict its occurrence in this case three days before the first convulsion.

In the following cases there was similar, sudden fever, running an acute course, but without any convulsions. In these, of course, the old theory of convulsive fever cannot be entertained at all.

Bouveret reports the case of a woman who came to the hospital with well-defined indications of chronic interstitial nephritis. There was considerable gastric irritability, vomiting, etc., and the temperature was 102.8° F. The following day the symptoms increased and the temperature continued elevated. This continued four days, when the temperature rose to 108.6° F., and the patient became comatose and died. At the time of death the thermometer registered 109.6° F., while an hour later it was 110° F. There was at no time any tendency to convulsion. At the autopsy there was found a high grade of contraction of the kidneys and some œdema of the lungs, together with hypertrophy of the heart, but no lesion of any other organ or structure including the brain cord and their membranes.

Richardiere and Therese record one case in their series having the clinical features of chronic interstitial nephritis, in which uræmia was suddenly developed in the form of coma, Cheyne-Stokes respirations, and sudden elevation of the temperature to 102.2° F. There were no convulsions, and the temperature remained between 102° and 103° F. for three days, when the patient died. Autopsy revealed the lesions of chronic interstitial nephritis, and there was some sub-arachnoid effusion of serum.

Chantemesse and Tenneson detail an interesting case occurring in a young woman of thirty-one years, who had had slight and transient œdema without albuminuria shortly before the delivery of twins. Eleven days after the labor there was pain in the brachial plexus and back of the neck, with large albuminuria and somewhat fetid lochia. During the next five days there were shifting palsies, mostly hemiplegic, loss of consciousness, dimness of vision, and ascending fever. The temperature finally reached 105.2° F. on the sixth day, and the patient perished. At the autopsy the kidneys were swollen and congested, and there was congestion of the membranes and substance of the brain and cord.

In a second case a man of sixty-six years who had had two previous attacks of unconsciousness became suddenly comatose, and showed diminution of sensation with loss of power on the right side, There was decided albuminuria. The temperature was 104.8° F. in the rectum, and later 106.2° F. At the autopsy arterio-sclerotic kidneys, atheroma of the aorta, some congestion of the bases of the lungs, and dropsy of the ventricles were detected.

In a third case the temperature rose during an attack of coma and hemilateral convulsions to 100.2° F., and remained between this point and 99° F. for over two weeks, during which the patient was more or less stuperous, but not convulsed. There was decided albuminuria. The patient passed from their observation shortly afterward.

A striking case of sudden uræmia, with delirium and high fever, terminating after free diaphoresis was observed by Damaschino.[15]

I have seen several cases of sudden uræmic coma with similar eclamptic fever, but have not preserved accurate clinical notes of any.

It will be seen from a study of the cases detailed that the temperature in this variety of uræmic fever rises more gradually than would at first seem to be the case. In nearly all, whether there are convulsions or not, there is an elevation for one or several days of a degree or two. When convulsions set in they are, as a rule, preceded by sharp rise of temperature to 102°, 103° or 104° F., and following the fit there is generally still more elevation, sometimes to 105°, 106° or 107° F. In fatal cases the high temperatures persist to the end, and there may be a further rise of a degree or more after death. In cases that are not fatal the temperature declines very soon and reaches the normal in a day or two unless the convulsions or other signs of the intoxication persist, when the temperature may also continue elevated.

II. The second form of febrile uræmia alluded to is that in which the onset is rather insidious, or at least not attended with the violent symptoms seen in the eclamptic cases, and in which the clinical manifestations are rather those of a slow infectious process not unlike those seen in the typhoid state. Systematic writers speak of such cases in reference to the differential diagnosis of typhoid fever (Osler[16], Pepper[17], Chantemesse[18]); but the literature does not furnish many accurately studied cases. In these cases the patient is frequently delirious, the tongue is dry and coated, the bowels constipated or relaxed, and the resemblance to an asthenic febrile disease like typhoid fever may be very close. The symptoms and the features by which the differential diagnosis is made will be better described in connection with the cases to be detailed.

A case of this kind, but without fever, was observed by Richardiere[19] in a young child of eleven years. The prominent symptoms in this were dropsy, stupor, delirium and headache, but there was no elevation of the temperature. Another case in which delirium occupied a prominent part in the clinical history of the case was recorded by Derignac[20], but this also was afebrile. Recently Josipovici[21] records a case of uræmia with the clinical features of an infectious disease, and Roch[22] published a contribution, to which I could not gain access, on the typhoid form of ursæmia. The following cases of my own, however, suffice to indicate the nature of this form of ursæmia.

Case VII.—T. P., aged seventy-three years, was admitted to my care, complaining of great heaviness and torpor. There was nothing of interest in his family history. He had had Chagres fever in South America many years before. Five years before the present illness he had an attack of unconsciousness, which lasted several hours. Since that time he had had frequent attacks of shortness of breath, sometimes very extreme, and cough, with frothy expectoration. There was also considerable epigastric soreness.

On admission the man was in a stupid condition, and there was considerable dyspnœa. The tongue was dry and covered with a brown fur; the bowels were a little loose; the pulse was slow and tense. Little urine could be obtained, but it was decidedly albuminous. On physical examination a rather high grade of pulmonary emphysema was determined, and there were besides soft, moist râles throughout both lungs. The heart-action was irregular, and the size of the organ difficult to determine on account of the emphysema of the lungs. The skin of the patient was dry and harsh; the temperature normal.

In the afternoon the patient began to wander in his mind, and there was slight fever. During the night there was very decided delirium, and the man could be kept in bed only with great difficulty. These symptoms persisted for two weeks, after which the patient died. The only new symptom was vomiting, which was noted one day. The prominent features of the case were the continuous delirium, the great depression, the restlessness, the dry-coated tongue, all of which gave the case a decided resemblance to typhoid fever on casual examination. There was, however, no enlargement of the spleen; there was no eruption of any kind, and the diarrhœa was not of the usual character met with in this disease. The pupils were small; the urine was constantly decreased in amount. Death occurred after two weeks of practically the same symptoms, apparently from exhaustion.

Autopsy. Emaciated old man; chest of the barrel-shape character. On opening the thorax the heart was seen to distend the pericardial sac, and on removal was found greatly hypertrophied, the wall of the left ventricle being two inches thick. There was no valvular disease of any kind. The heart weighed twenty-two ounces; the lungs were slightly œdematous; the kidneys were small, rather light in color, and the left contained a small retention cyst. On section the cortical substance was found to be of a light color and seemingly very fatty; the vessels in the kidney substance were patulous and stiff. The capsule was somewhat adherent. Weight of the kidneys: left, three and one-half ounces; right, six ounces.

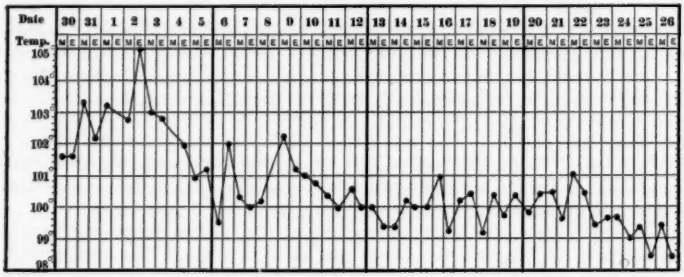
The temperature in this case was never high, though on several occasions it passed 99° F.; but the other symptoms were so decidedly typhoidal that I venture to include it in the group of slow febrile uræmia.

A second case is more striking and deserves to be reported in detail:

Case VIII.—Mrs. S. was admitted to the gynecological wards as a supposed case of sepsis on July 30, 1894. She passed through a normal labor two months previously, and seemed to have recovered when the present condition of fever and depression was developed. On admission to the hospital the temperature was 101.6° F., the respirations 34, and the pulse 100; the patient was in a stuperous or almost comatose condition, there was occasional twitching of the hands and feet, especially on one side, and the body was covered with an eruption of sudemia and miliaria. The tongue was coated but moist. Physical examination revealed a rather harsh form of inspiratory murmur, but no other indication of pulmonary disturbance. The pulse was quite tense, and the second aortic sound was accentuated, but there was no murmur. The pupils were about normal in size and somewhat sluggish in action; the abdomen was flat—there was no pain or tenderness; the spleen was

not enlarged; the bowels had been costive for several days. There was a urinous odor about the patient which was not decided, but still suggestive. The urine was scanty and had a specific gravity of 1018; it contained abundant albumin and hyaline and granular casts in very large numbers. The provisional diagnosis of uræmia was made, and the patient was ordered Rochelle salts in hourly doses of a drachm each, with the result that she had twenty large, watery stools in the next twenty-four hours. Very soon the stuperous condition cleared away, the pulse softened, the respirations grew more regular and natural in sound, and the twitching ceased entirely.

CHART III.



Further examination of the patient was now made. The blood was found to contain 4,300,000 red corpuscles and 17,400 leucocytes. The stained blood showed about the same proportion of lymphocytes, large and small, polynuclear elements and eosinophilous forms in a count of several hundred leucocytes.

On vaginal examination a contracted, hard, and slightly retroverted uterus was discovered, and there was no vaginal discharge. Abdominal examination still continued negative. The urine still presented the same large albuminuria and abundance of casts. The temperature was kept under control by sponging, which was repeated twenty-three times in the first three days. After this time no more sponging was needed. With the improvement in the patient's general condition there was a subsidence in the rapidity of the pulse and a fall in the temperature. After the fourth day the former fell to between 60 and 80 in the minute. The respirations continued rapid, but were far more natural in character. There was no more twitching. On August 10th there appeared a painful swelling of the calf of the left leg. This extended upward along the line of the femoral vein, which could be felt as a hardened cord. There was some slight coldness and blueness of the foot, but under treatment by rest, elevation, and sedative applications, all symptoms subsided after a few days.

The patient's general condition improved immediately after the first thorough purgation, and still more after the administration of digitalis, beginning August 3d. The albumin gradually decreased in amount, and was sometimes absent entirely. When the patient was finally discharged there were neither casts nor albumin. The urine was examined for pepton with negative result. The blood at the second examination

showed 3,300,000 red corpuscles and 31,200 leucocytes, with 70 per cent. of hæmoglobin.

Clinically this case suggested three possibilities: typhoid fever, puerperal sepsis, and uræmia. The diagnosis seemed to me to rest with the last condition. The appearance of the patient at once, however, suggested typhoid fever, and so strikingly that it was more difficult to eliminate this than the clinical report would indicate. The absence, however, of splenic enlargement, of intestinal symptoms, of rose spots, and the presence of distinct leucocytosis were points of considerable importance in the diagnosis. Of course the possibility of primary typhoid fever with secondary nephritis and consequent uræmia made the evidences of uræmia, that were unmistakably present, of no value in the exclusion of typhoid fever. The frequency of phlebitis and thrombosis in typhoid fever and their infrequency in nephritis would, of course, have some value in the diagnosis. I find, however, in the records of Prof. Tyson, at the University Hospital, a case of pure nephritis in which femoral thrombosis and gangrene developed while he was under observation in the hospital. The points indicated, however, served to set this disease aside, and the subsequent history of the case was confirmatory. The question of puerperal sepsis was perhaps even more troublesome. There was not, it is true, any evidence of gross infection with uterine or parauterine disease, since the gynecologist reported the uterus and its appendages normal; but the possibility of infection without marked local reaction, and with particular or sole involvement of the kidneys, as in some of the reported cases of infectious nephritis, required careful consideration. It was to be remembered in the first place that the diagnosis was concerned with the state of the patient at the time of her entrance into the hospital rather than with the determination of the original cause of her disease. There was undoubtedly a condition of acute nephritis, possibly of infectious origin; but the question to be decided was whether the associated symptoms were due to uræmia or to infection, no matter whether the primary nephritis were infectious or not. My opinion of this matter was very promptly formed. The woman was undoubtedly uræmic, as the condition of the mind, the twitching, the odor of the patient's skin and breath, and the scanty albuminous urine indicated. The effect of treatment served only to strengthen this opinion, for almost at once, after the first thorough purgation, the stupor, twitching, and odor disappeared, as might be expected in uræmic conditions, but scarcely in pure sepsis. I say nothing of the subsidence of the temperature, though I regard this also as significant, for the reason that it is my present purpose to establish the significance of fever in uræmia, rather than to form deductions from its presence or absence. After the first decline of the fever and subsidence of the uræmic symptoms it will

be noted that there was a continuance of a milder degree of fever. This may have been due to lingering uræmic intoxication or to the continued existence of infection or of an inflammatory condition of the kidneys. It seems to me not in the least unlikely that the last assumption may have considerable foundation, but it is impossible to settle the question definitely. The earlier typhoidal symptoms, however, seem undoubtedly to have been uræmic, since they disappeared so promptly and with such exact correspondence to the degree of purgation; the substratum of infectious nephritis, with the fever proper to it, would not be influenced in the same prompt and decided manner, and, would therefore, remain after the removal of the uræmic element. The idea of sepsis entering even into the causation of the nephritis itself seems to me to be negatived by the fact that the labor was normal and had preceded the first indication of kidney trouble by fully two months; and still more by the fact that the patient presented none of the usual indications of sepsis.

SUMMARY.—Fever may occur in the course of Bright's disease under three conditions: 1. At the onset of acute nephritis as a result of the infection or intoxication causing the disease, or of the inflammatory or degenerative lesions in the kidney themselves; 2, in the course of acute or chronic Bright's disease as a result of various complications; and, 3, in uræmia. The fever associated with uræmia may be sudden and pronounced, and associated with coma, delirium, or convulsions — eclamptic fever; or it may be more general, more lasting, less marked, and associated with typhoidal symptoms—slow uræmic fever.

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RETRO-POSITION OF THE UTERUS.

By George W. Jarman, M.D.,
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Although the writer uses the word retro-position in the title of this paper, he is convinced that it is not the term which most correctly designates the condition under consideration.

Nearly all authors who describe this displacement of the uterus use the term retro-position to express not only this, but another malposition, namely, retroversion, which has nothing in common with the one under consideration.

It is needless to say that this usage of a term to describe two very different positions of the uterus can result only in confusion. The writer would use the word retrocession as expressing more perfectly the real position than retro-position. Retrocession carries with it no idea of any turning of the uterus around a transverse axis. It simply means that the uterus has receded toward the sacrum.

Schultze, Winkel, Hart and Barbour, and Pozzi, all speak of this malposition, yet in so meagre a manner as to leave one to suppose that it is not only very infrequent, but that few symptoms accompany it. From an experience extending over five years in one of the largest gynecological clinics in this city, the writer has found that retrocession of the uterus existed in the proportion of one to eighteen, where malposition of the uterus was diagnosed, and that symptoms so marked depended upon this position that treatment was desired.

The writer bases the deductions in this paper upon his observation of sixty-two patients with retrocession.

Inasmuch as the utero-sacral ligaments and the peritoneal surfaces of Douglas's pouch are so important in the production of this displacement, it will be necessary to make slight reference to their anatomy.

"The utero-sacral ligaments are peritoneal folds, two in number, inclosing connective tissue and unstriped muscular fibre, passing from the lower lateral part of the body of the uterus outward and backward toward the second sacral vertebræ" (Hart and Barbour). They form the lateral boundaries of the Pouch of Douglas. The sacral attachments of these ligaments is on a higher plane than their uterine ends, and if they become shortened the uterus is not only drawn backward, but is also lifted upward. The lymphatics of the vagina, cervix, and uterus are very numerous and complex, and it seems almost impossible, from an anatomical standpoint only, to determine their distribution. According to Sappey, the lymphatics of the upper three-fourths of the vagina and cervix enter the hypogastric glands which lie subperitoneally

in the space between the external and internal iliac vessels. Le Bec, however, holds that the lymphatics from these structures pass out underneath the broad ligaments to the obturator gland of Guérin.

Recognizing how very rich the pelvic organs are in lymphatics, and bringing to bear the assistance of clinical observation, it would seem that it is not paradoxical to state that both authorities are right. We all see instances of inflammatory exudate following laceration of the cervix with septic infection, where the extension is sometimes in the broad ligament, and again retrouterine. This indeed seems to offer the true explanation of the inflammation which is found to exist in the utero-sacral ligaments. The cervical mucous membrane becomes inflamed,

FIG. 1.

Diagram of vertical mesial section of female pelvis, showing normal distance between uterus and sacrum. Bladder empty.

and septic material travels through the lymphatics which lie underneath the peritoneal covering of these ligaments, and they in turn become inflamed. This process may go on so slowly that the patient will complain only of backache, and, attributing it merely to overexertion or to some other possible cause, may not even seek treatment. It is only after secondary contraction, which follows this new infiltration of connective tissue, takes place that the real condition of the retrocession exists. (See Figs. 1 and 2, showing the normal and retroceded uterus.)

Should the patient possess an unusually long cervix, the condition of pathological anteflexion will be induced by the contraction of these

ligaments. This is perfectly easy of explanation when we consider that the uterus is constantly subjected to intra-abdominal pressure which tends to force the fundus of the normally placed organ forward toward the pubes, while the contractile power of the vagina tends to force the cervix in its own axis, which is nearly at right angles to that of the body of the uterus. Hence, should the cervix be abnormally long, the leverage will be increased so that it must assume the axis of the vagina.

It is the writer's belief that it is simply because operators have overlooked this retrocession and have given their entire attention to one of its frequent effects, namely, anteflexion, that such poor results have

FIG. 2.

Diagram of vertical mesial section of female pelvis, showing contracted utero-sacral ligament, with uterus in the position of retrocession.

been obtained from the treatment of so-called cases of anteflexion. The recent operation devised by Dr. E. C. Dudley in reality shortens the intravaginal portion of the cervix, hence decreases the leverage, also affords free drainage for the endocervicitis, and it seems quite possible that this would explain, at least in some of the cases, the marked relief obtained. On the other hand, viewing the subject from the standpoint that the retrocession is so frequently the cause of the anteflexion it is not surprising that failure to relieve the symptoms so often follows even extreme divulsion and tamponade. It has been said above that the uterus is not only drawn backward by reason of the shortened utero-

sacral ligaments, but that it is also lifted upward, nearer to the brim of the pelvis, inasmuch as the sacral attachment is on a higher plane than the uterine.

This mechanical elevation of the uterus deprives it of the natural support offered by the floor of the pelvis. In this way these already inflamed ligaments are forced to sustain more weight than when the uterus is in its normal position. The utero-sacral ligaments receive their nerve supply, in common with the uterus, through the sympathetic system. Branches from the hypogastric plexus, which lies between the common iliac arteries, reinforced by branches from the lumbar and sacral ganglia and sacral nerves, form the inferior hypogastric plexuses, and from these filaments pass to the utero-sacral ligaments.

Pain in the sacral and lumbar regions is the most constant symptom associated with retrocession. This pain is often described by patients as being similiar to that which they suppose they would experience if the back was broken, their expression being a "back-breaking pain." Standing, or in fact whenever the patient assumes the upright posture, increases this pain. It would be difficult to understand why this should not be the case when we remember the increased weight which the ligaments are called upon to support. Women who are compelled to do their own work and lift heavy weights will suffer more than those who can assume the recumbent posture more frequently. Again, any constriction around the waist of patients with this malposition will intensify this pain by increasing the intra-abdominal pressure. Sterility existed in nearly all the patients who came under the writer's observation. That this condition is due to the position of the uterus is not claimed, but rather that the most frequent cause of the inflammatory process in the utero-sacral ligaments, namely, endocervicitis, seems the most plausible explanation of the sterility.

Vesical irritation is another symptom which accompanies this displacement. That this should be the case can readily be understood, if the changes in position of the normal uterus depending upon the contents of the bladder be borne in mind.

When the uterus has receded from its midway position between pubes and sacrum, it must of necessity render its anterior attachment more tense.

The vagina will be found unusually deep and both anterior and posterior walls overstretched.

If the cervix uteri is long, it will be found in the axis of the vagina and forming an acute angle with the body of the uterus. Any attempt to move the uterus will cause the patient pain, which she refers to the back. Making traction on the cervix with a tenaculum, with the examining finger in the posterior fornix, will reveal the fact that the uterus has lost its normal mobility, and at the same time the tense utero-sacral

ligaments can easily be made out. They will appear as two diverging cords attached to the posterior surface of the lower uterine segment, passing upward, backward, and outward. The uterus is frequently found to be in marked anteversion, caused by the round ligaments drawing the fundus forward. It is almost impossible to mistake this condition for any other.

Any inflammatory exudate in the cul-de-sac of Douglas may simulate retrocession, but the uterus will be more firmly fixed and will not be so near the sacrum. Spasmodic contraction of the ligaments of the uterus, a condition not infrequently observed in hyperaesthetic patients, might possibly be mistaken for retrocession. An examination under chloroform would reveal the true condition.

Inasmuch as the condition which produces retrocession of the uterus is chronic, the non-operative treatment must be prolonged, if satisfactory results are to be obtained.

The cervical canal should be examined and the cervical endometritis relieved. Thorough curettage under local anæsthesia offers the best means for rapidly disposing of this condition. If the cervix is abnormally long it should be amputated. Lifting the uterus up, so that the strain on the utero-sacral ligaments is lessened, will relieve the patient's symptoms to a great extent. This may be done either with the tampon suitably medicated or else with the retroversion pessary. It is not always possible to use the latter until after the tenderness of these ligaments has been reduced. The writer has not seen any good results from hot douches in the treatment of these cases. Chloral hydrate solution in glycerin, in proportion of two parts to twenty, applied by means of the tampon, having a distinct analgesic effect, has relieved the pain of which these patients complain more certainly than any other local treatment.

Massage is one of the surest methods of curing the patient of retrocession. I do not mean in this instance the ordinary pelvic massage, but the actual stretching of the contracted ligaments. It will often be necessary to restrict treatment to less harsh methods until the local tenderness is somewhat abated. As soon as the patient will bear it, and selecting a time at least four days distant from the menstrual period, it can be employed. The vagina, cervix, and cervical canal should be rendered as aseptic as possible, and with a double tenaculum the anterior lip of the cervix can be grasped. This procedure in reality produces very little pain on account of the insensitiveness of the cervix. Making traction on the tenaculum, with the examining finger of the other hand in the posterior vaginal fornix, will put these shortened ligaments on even greater tension and permit the operator to press his finger against them both transverse and laterally. The amount of force which can be exercised will be limited by the degree of pain

which is produced, it being advisable to stop short of severe pain. Gentleness and persistence will accomplish far more than harsh handling. It is needless to say that in this, as in other forms of pelvic massage, the operator must be careful not to let his hand come in contact with the vestibule or clitoris.

Although it is not possible for the writer to prove the statement, yet clinical experience has demonstrated to his own mind that the red iodide of mercury, administered internally, has been of marked benefit to patients with retrocession. The only explanation possible is that it stimulates the lymphatic system and causes it to take up the products of the inflammatory process. At any rate he has made use of it in quite a number of cases, and has satisfied himself that it has been at least an adjuvant in the treatment of retrocession.

The bowels should be kept active. Electricity has decided pain-relieving power in these cases and should not be overlooked. Where, from lack of time or the impossibility of securing the regular services of a physician, local treatment becomes impossible, or where such treatment fails to bring about the desired relief, operative methods will be advisable.

Since the revival of the vaginal operation for the relief of abnormal conditions of the pelvic organs, it has been demonstrated that these ligaments can be cut with perfect safety.

In performing vaginal hysterectomy, as soon as these ligaments are divided, the increased mobility of the uterus is marked. The same thing is found when doing this operation by the abdominal method; the uterus can often be raised into the abdominal incision after its posterior attachments have been liberated, whereas previous to this procedure the organ may have appeared almost fixed.

There can be almost no danger in the operation for the division of the utero-sacral ligaments through the vaginal route. Under strict antiseptic precautions a transverse incision, one inch in extent, through the posterior fornix, can be made and Douglas's pouch opened. Traction on the cervix will render the ligaments tense and their location simple. With the guidance of the index finger their anterior attachment can be cut with blunt-pointed scissors. Hemorrhage from this source would be slight and under perfect control. The traction of the vaginal walls is sufficient to hold the uterus forward during the process of recovery. The incision through the fornix could be closed with sutures, as is done following vaginal ovariotomy. The writer had an opportunity of witnessing a very recently similar procedure in the hands of Prof. Henry C. Coe. In this case there was a small fibroid immediately behind the uterus, apparently attached to one of the utero-sacral ligaments. During the process of its removal it was necessary to cut these ligaments. As soon as they were divided the uterus sprang forward to its normal relation-

ship with the pubes. It had been in marked retrocession. The case demonstrated the ease of access to these ligaments through the posterior vaginal vault.

The writer would draw the following conclusions:

1. That retrocession has been too often overlooked in the explanation of pelvic pain.

2. That the condition of the utero-sacral ligaments must be taken into consideration in the treatment of anteflexion.

3. That this condition can easily be relieved by operative measures, if local treatment does not succeed.

AN INVESTIGATION INTO THE SO-CALLED LYMPHOID NODULES OF THE LIVER IN ABDOMINAL TYPHUS.

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The pathological changes met with in the liver of those who have died of typhus abdominalis have, for a long time, attracted the attention of investigators. Perhaps to Friedreich[1] should be given the credit for the discovery of certain circumscribed nodular formations which he described as occurring in the liver of a girl, twenty-one years of age, who had died of typhoid fever. He found, in the neighborhood of the coarser bands of stroma, collections of cells which could not be seen with the naked eye, but which, viewed microscopically, consisted of round and partly irregular cells imbedded in the connective tissue of the organ. While this description is not complete, since Friedreich only saw these collections of cells in the interstitial connective tissue, yet it is, we think, sufficiently accurate to justify the belief that he saw what E. Wagner,[2] three years later, so minutely described. The latter's classical description of the hepatic lesions seen by him in five patients who had died of typhus abdominalis has scarcely been added to up to the present time. As regards his first case, Wagner describes the liver as of normal size and anæmic in appearance, somewhat softened, and with the outlines of the lobules obscure. He saw over all portions of the organ quite numerous, small areas, which were just on the limit of visibility, of a gray color, and fairly solid in character. This refers to the naked-eye appearance. Upon microscopical examination these spots were found to be round, oblong, sometimes irregular, serrated and

[1] Ein neuer Fall von Leukamie, Virchow's Archiv, 1857, Band xii. S. 53.

[2] Beiträge zur pathologischen Anatomie der Leber bei Abdominal Typhus, Archiv d. Heilkunde, 1860, Band i. S. 322.

biscuit-shaped, the majority of them occupying the actual parenchyma of the organ, and replacing the hepatic cells. Now and then they invaded the interlobular connective tissue. They consisted of closely lying groups of small, round and oval refractive nuclei; in some places, on the periphery of the area masses of these nuclei, of pointed shape, projected into the surrounding liver tissue. Wagner was of the opinion that these nuclei were derived partly from the cells of the interacinous connective tissue, and that in part they had their origin within the lobules. He was unable to completely demonstrate the manner of production last mentioned, but thought that these nuclei owed their origin to the nuclei of the liver cells. In a second case of typhoid fever, Wagner[1] saw these fine, barely visible, sharply circumscribed grayish specks over all parts of the cut surface of the liver. As a rule they were immediately on the periphery of the small portal veins, and consisted of albuminoid molecules between which small round refractive nuclei were quite regularly distributed, and in contrast with the central part of the area, which appeared to consist of irregular particles, the periphery was largely made up of round nuclei.

The third case investigated by Wagner[2] is of interest in connection with our own investigations. The patient had recovered from an attack of typhoid fever, but had died from some other disease two and a half months later. The characteristic nodules were not found in this case, and the inference is drawn that they had been present, but had entirely disappeared, their place being probably taken by a new formation of liver cells.

Hoffmann,[3] referring to the previous investigations of Wagner, states that he has also found in a considerable number of cases of typhoid fever small grayish nodules in the liver, and says that in most cases it requires close inspection in order to see them with the naked eye; that in some cases they are quite distinct, and on the cut surface, appear as flat elevations; microscopically they appear as round conglomerations of small round cells and nuclei, resembling the lymphoid nodules found in the peritoneum. He states that their location is, in most cases, within the lobules, and close to one of the small portal spaces. He found, almost without exception, a diffuse infiltration of cells which followed the ramifications of the portal vein. He regards them as lymphoid cells which have wandered in from the portal vein. Of 250 cases examined, he observed these nodules in the liver in 38, and in 14 of the 38 the nodules could be seen with the naked eye.

[1] Die Körnchenbildung in der Leber, Arch d. Heilkunde, 1861, Band ii. S. 103-114.

[2] Beiträge zur Pathologie und pathologischen Anatomie der Leber, Deutsches Archiv für klin. Med., 1883-4, Band xxxiv. S. 520-537.

[3] Untersuchungen über die pathologisch-anatomischen Veränderungen der Organe beim abdominal Typhus, Leipzig, 1869, S. 221-222.

Orth[1] states that the microscopic gray nodules found in the liver of typhus abdominalis are of the same formation as those seen in leukæmia and lymph adenomata.

Fraenkel and Simmonds,[2] while studying the hepatic changes in abdominal typhus, always found lesions which they considered to be intimately connected with the disease, viz., small cell infiltration of the connective tissue, and those peculiar formations generally known as lymphomata. They describe two forms which they think are deserving of the latter term: First, those areas which consist of closely packed small, round cells, amongst which may be recognized, here and there, liver cells. Secondly, those in which the round cell accumulation is less close, and which appear to consist largely of the remains of poorly staining liver cells. There are also small, circumscribed areas of liver tissue, in which the protoplasm and nuclei of the cells only stain imperfectly, or not at all, and which do not deserve to be designated as lymphomata. They are inclined to consider this latter form as one of coagulation necrosis. They are further of the opinion that in the beginning the change is one of circumscribed degeneration, and that later there takes place an accumulation of round cells within the necrotic area. They regard as improbable any direct connection between the presence of typhus bacilli and these formations, although they once found a clump of the bacilli within an affected spot.

Handford[3] states that in all fatal cases of typhoid fever examined by him there were found definite changes in the liver, in the form of parenchymatous and interstitial changes. The most characteristic change, though not constant, is the presence of small, round areas that stain imperfectly, that are crowded more or less thickly with leucocytes, and surrounded by a dense ring of cellular infiltration. The liver cells in the patches show cloudy swelling or more advanced degeneration. In more advanced stages the liver cells cannot be distinguished at all, and mingled with the leucocytes are irregularly shaped and spindle cells. In fact the tissue resembles an early stage of cicatricial tissue. In a third variety the patches cannot be distinguished from miliary abscesses. Handford regards the last as a septic hepatitis, due to absorption of septic particles from the intestine, while the former are due to capillary embolism. He is not satisfied that any specific bacillus is the cause of these lesions.

Siredey[4] states that during the course of the second week, and sometimes during the third week of typhoid fever, the microscopical appear-

[1] Lehrbuch der speciellen pathologischen Anatomie, 1887, Band i, S., 954.

[2] Die aetiologische Bedeutung des Typhus Bacillus, Leipzig, 1886.

[3] "Hepatitis in Enteric Fever." Transactions of the Pathological Society of London, 1889, pp. 129–132.

[4] Contribution a l'études des alterations du foie dans les maladies infectieuses, Rev. de Méd. vi., Paris, 1886.

ance found on section of the liver is so peculiar and distinct that one can, in the majority of cases from these appearances alone, make a diagnosis of that disease. He describes the lesions as being scattered in various parts of the hepatic substance, and says that their form, location, and size are extremely variable; that while ordinarily round, they are sometimes ovoid or even star-shaped. They are oftener met with at the centre and in the midst of the lobule than at the periphery. He remarks that they offer the greatest analogy in their general shape to tuberculous nodules; but they do not present the distinct layers found in the latter, nor does one see giant cells, nor epithelioid cells. He states that he has never seen any tendency to caseation in these nodules; that they never include hepatic cells, and that the cells composing them are but little, if at all altered. He further says that although these nodules are perhaps special to typhoid fever, and that when once encountered in the liver the diagnosis is probable, yet there are other infectious maladies characterized by the migration of lymphoid cells into the liver, and that these can under certain circumstances present themselves as nodular conglomerations. He expresses the probable opinion that after convalescence the liver parenchyma is fully restored to its former integrity without any lesion being left behind.

Legry[1] has contributed an interesting monograph to this subject. In addition to a study of the lesions found in the human liver in typhoid fever, he has injected pure cultures of typhoid bacilli into the mesenteric vein of rabbits, and afterward investigated the change, if any, found in the liver. The fact is worth mentioning in this connection, that our experiments on rabbits, carried out in much the same manner as Legry had done, were completed prior to our acquaintance with his monograph, and that while his experiments were attended with negative results as far as finding any changes in the rabbit's liver are concerned, our own experiments were followed by characteristic lesions, as will be later shown. He gives much the same general description as to the location of these nodules as Siredey had already done; but does not agree with him that they consist of lymphoid cells. He supports the conclusion that these nodules, which are found most often along the portal spaces, are always formed of nuclei, generally abundant, surrounded by a granular substance, which latter appears to result from the degenerated protoplasm of liver cells. He gives no definite opinion as to the source of these nuclei, but thinks that the nodules have a bacterial origin. He never found the bacilli within the nodules, but has found upon section, six times in eleven cases, characteristic clumps of typhoid bacilli. His experiments upon rabbits (two cases) gave no definite results.

It will thus be seen that each of the writers to whose investigations

[1] Contribution a l'étude du foie dans le fievre typhoide. Paris, 1890.

we have referred describe, with more or less particularity, certain nodular formations found in the liver of those who have died of typhoid fever.

Our own investigations have been carried on in the Pathological Laboratory of the Johns Hopkins University, and have consisted in a careful study, microscopically, of the livers of five patients who had died of typhoid fever in the wards of the Johns Hopkins Hospital. We have also injected pure cultures of typhoid bacilli into the mesenteric vein of rabbits, and afterward studied the lesions found in their livers.

Before we proceed to a description of the microscopic appearances we wish to state that although it is not always possible to distinguish with the naked eye the nodular formations which Wagner discovered microscopically, yet a patient search of the cut surface of the organ will generally be rewarded by finding here and there very small, roundish, grayish white specks within the lobules; sometimes they are quite abundant and easily to be seen—again just on the limits of visibility, and with difficulty found.

Microscopic Appearance of the Liver. In two of the cases studied by us frozen sections of the fresh liver were made. In addition to the well-marked fatty infiltration of the cells, the latter being generally filled with large fat drops, there were also to be seen within the lobules well-defined areas which contained many fatty granular cells situated in the midst of a coarse granular material. Within these areas there were also to be observed many irregular-shaped nuclei.

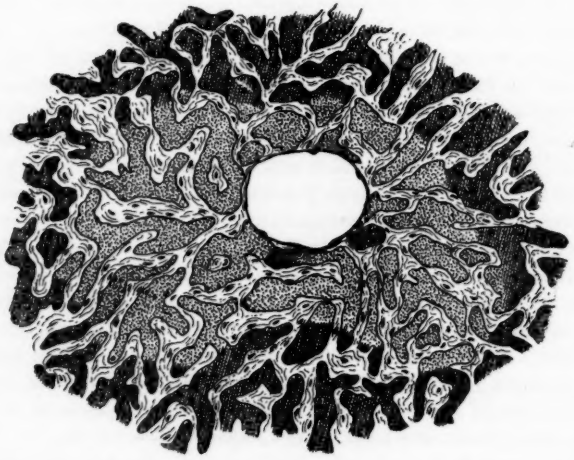
For the further study of the microscopic appearances the following method was used, viz.: Fresh portions of the liver were hardened in 95 per cent. alcohol, and afterward imbedded in paraffin or celloidin. Sections of these were then stained with Löffler's methylene-blue solution for ten to thirty minutes; then transferred to ½ per cent. aqueous solution of eosin, where they remained from fifteen to forty-five seconds; next decolorized and dehydrated in absolute alcohol, cleared in oil of cloves or bergamot, and mounted in Canada balsam.

If, as is sometimes the case, we are unable to see with the naked eye the small whitish areas on the cut surface of the liver of typhoid fever, when we come to the microscopical examination of the organ we no longer experience any difficulty in finding them. If one has succeeded in obtaining a properly stained section a most striking picture is presented to the eye when examined with the low power, and that feature which will most quickly fix the attention is the presence of bright, sharply defined eosin-stained areas of various shapes and sizes, which are to be seen scattered plentifully throughout the section, and in almost every field. Sometimes these areas are almost perfectly round, more often oval, and generally irregular in outline. The sizes of these foci are as variable as their shapes, embracing now only a few liver cells, again involving as much as one-half or even the whole of a lobule. Their

relative position is also very variable; sometimes we find them near the portal spaces—again in the midst of the lobule, and quite as often situated near the central vein. We have several times observed an area occupying the entire periphery of the central vein. It cannot be said, we think, that these areas stand in closer relation to the portal vein than to the hepatic spaces. If the section has been well stained there should never be any difficulty in picking out even the smallest necroses, since the contrast between the healthy cells, which are stained with methylene-blue, and the necrotic eosin-stained cells is always well marked.

The appearance of these areas is variable. Sometimes we see a spot involving as much as one-fourth of a lobule in which the liver-cells have

FIG. 1.



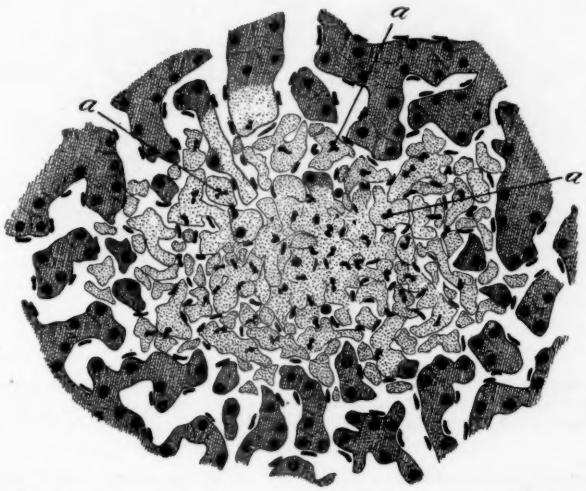
A necrotic area of the human liver of typhoid fever. The area of necrosis entirely surrounds the central vein of the lobule; the cells have lost their nuclei, but still retain their continuity with the healthy cells. Drawn with camera lucida, Bausch & Lomb 1/6 objective, Leitz ocular No. 4. Reduced ¼.

apparently died *en masse*, but continue to retain their shape perfectly; even the demarcation between the individual cells can still be plainly made out. The beam-work of the cells will not be at all broken, but will be seen to be continuous with that of the surrounding healthy cells (Fig. 1). The capillaries of such an area will be found to be unaltered, or perhaps slightly dilated. Continuing the examination with the low power, we will most often fail to discover any nuclei whatever within the finely granular eosin-staining cells; and even with a high amplifica-

tion may only find within these cells minute particles of nuclear detritus. We have seen several such areas as the one now being described, in which there appeared no increase whatever of the cells within the capillaries situated between the rows of necrotic cells. In other instances we have found the capillaries to contain a considerable number of polynuclear leucocytes. Sometimes we have seen a marked increase of polynuclear cells within the capillaries, near the centre of the area; again, the largest number will be found about the periphery of the necrosis. We are inclined to regard the areas just described as probably the earliest form of the local parenchymatous degeneration. These are the areas which Frankel and Simmonds consider to have undergone coagulation necrosis, and which, in their opinion, do not deserve to be designated as lymphomata.

Far more frequently one will see areas wherein the liver cells no longer preserve the regularity of their framework, but have become dis-

FIG. 2.



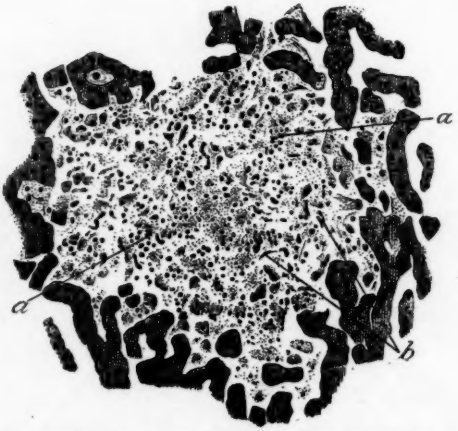
Necrosis of human liver in typhoid fever. *a*. Polynuclear leucocytes within the dead hepatic cells, which latter have in large part lost their shape. Camera lucida drawing, Leitz 1/12 immersion, No. 3 ocular. Reduced ¼.

placed and have lost their shape. Some of these cells may still show pale and swollen nuclei, but the majority will have entirely lost their nuclei, and in their stead there will be discovered within many of the necrotic cells one or several polynuclear leucocytes (Fig. 2). The presence of the irregular and club-shaped nuclei within the necrotic cells of the area is one of the most striking and important features to be

observed. Although certain areas may show only a small number of such nuclei, as a rule they are found in great abundance. We have never, however, observed such an abundance of polynuclear leucocytes within a necrosis in the human liver as to give the picture of a miliary abscess such as is described by Hanford. Sometimes we find included within such an area, and completely surrounded by the necrotic cells, from one to a dozen or more apparently healthy liver cells which still preserve their nuclei, and which do not stain with eosin. These cells, as a rule, contain many fat drops, but in this respect do not differ much from the cells throughout the section.

In addition to the two varieties of areas described we will find others which have undergone yet further changes. Here the hepatic cells have

FIG. 3.



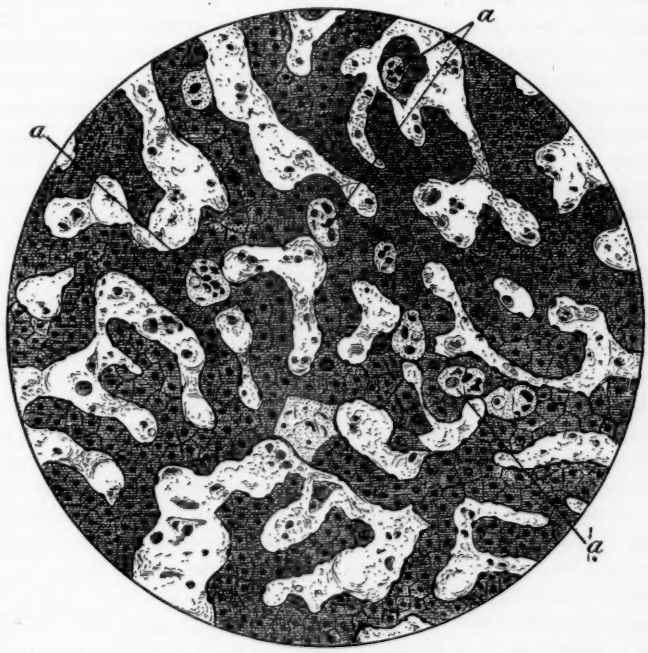
Later stage of necrotic area of human liver in typhoid fever. The cells have entirely lost their shape; many small round nuclei within area (*a*), and here and there a few spindle cells (*b*). Camera lucida drawing, Bausch & Lomb 1/6 objective, Leitz ocular No. 3. Reduced ¼.

quite disappeared, and are replaced by numerous small, round, deeply-staining nuclei, which crowd the area in all parts, intermixed with fewer oval, spindle-shaped nuclei (Fig. 3). In other words, the appearance is that of young connective-tissue, as mentioned by Hanford.

Besides these larger areas of necrosis, we wish to invite attention to the smaller necroses which are found in the liver. We here refer to the necrosis of individual liver-cells. We have found these smaller necroses in each of the cases examined. In one case they were a prominent feature of the examination (Fig. 4). We find scattered here and there amongst the healthy cells of the lobule one or more cells whose proto-

plasm stains brightly with eosin, and whose nuclei have, as a rule, entirely disappeared. We will generally observe that such necrotic cells contain within them several irregular-shaped nuclei, which plainly mark the entrance of polynuclear leucocytes into them.

FIG. 4.



Necrosis of individual cells in human liver, typhoid fever. At *a* are seen one or more cells which have partially or entirely lost their nuclei and which contain polynuclear leucocytes. In this section the capillaries were much dilated. Camera lucida drawing, Leitz 1/12 immersion objective, No. 3 ocular. Reduced ⅓.

RELATION OF BACILLI TO AREAS OF NECROSIS. We have found characteristic clumps of typhoid bacilli upon section in each of the five cases examined by us; it was generally possible, by careful search, to find one or more colonies in nearly every section, the bacilli lying always within the capillaries. As to the relation existing between these clumps of bacilli and the necrotic areas, we have examined a great many sections from each case, but have only succeeded in finding the bacilli within the areas five times. Four times the colony lay just within the margin of the area; in the remaining instance we observed a capillary

distended to twice its normal size by a large colony of bacilli, which was surrounded on all sides by necrotic liver-cells, so that the clump of bacilli occupied in this case the centre of the necrosis. We were at first inclined to believe that the finding of the colony of typhoid bacilli within the necrotic area was sufficient reason for assuming that the death of the liver-cells was due to the presence of the bacilli, or rather that the toxalbumin produced by the latter caused the death of the cells in their immediate vicinity. When, however, we had examined very many areas in which no bacilli could be discovered, and when we found colony after colony surrounded by liver-cells, which, to all appearances, were quite healthy, we were reluctantly compelled to leave in abeyance so plausible a theory. With the object, if possible, of coming to a final decision in this matter we made careful serial sections in two of our cases, but prolonged and careful search failed to discover any bacilli within these areas.

Considerable interest must attach to the final disposition of these localized necroses of the liver in those who have recovered from typhoid fever. One is struck with the large number of necrotic cells which must necessarily be removed and whose places must be filled by other cells. Siredey makes the rather surprising statement that during the third week of the disease these nodules no longer appear in the liver; as he believes that they consist largely of lymphoid cells which have been brought to the liver through the portal circulation, he supposes that by the third week they have already re-entered the general circulation. This supposed disappearance of the nodules during the period above mentioned is not borne out by observation, as we have found these lesions in the human liver as early as the second week of the disease, and as late as the seventh week after the attack had begun. Wagner states that they probably entirely disappear, and suggests a restoration of the tissue through a proliferation of the liver cells. While our investigations were being carried on we were so fortunate as to be able to examine microscopically the liver of a patient who had experienced an attack of typhoid fever about twenty-five years prior to death. This was the case of a woman aged about seventy years, who died of carcinoma of the ovary and was autopsied in the pathological laboratory of the Johns Hopkins Hospital. The clinical history of a former attack of typhoid fever was fully supported by finding in the lower ileum several large cicatrized areas, with raised, uneven edges. Microscopical examination of the sections taken from these old cicatrices showed that an ulcerative process had destroyed the mucosa in its entire thickness, and had extended down to the muscular layer. No muscularis mucosæ was to be seen; each ulcer had occupied the greater part of a Peyer's patch, and it was extremely interesting to observe that the follicles of Lieberkühn had been somewhat reproduced, these appearing

as much atrophied follicles resting directly on the muscular layer. Equal interest attached to the microscopical examinations of the hardened sections of the liver, for, aside from the general atrophy of the liver cells (the patient was quite old), there were to be seen in almost every field from one to three circular, round, ovoid, or irregular-shaped areas of dense connective tissue. These were, as a rule, sharply defined, being situated sometimes near the portal spaces, again around the hepatic vein, and often *within the lobules;* so that as regards relative position they corresponded to the areas of necroses found in the liver of typhoid fever. Within many of these old areas of connective tissue, blood-vessels and bile-ducts had been reproduced. While it would not be well to attach too much importance to the localized areas of connective tissue found in the liver in this case, we yet consider it of extreme interest to find such an unusual form of cirrhosis in the liver of a patient who had so clearly passed through a former attack of typhoid fever.

Lesions in the Livers of Rabbits Inoculated with Pure Cultures of Typhoid Bacilli.—Blackstein[1] failed to discover any lesions in the liver of rabbits into whose circulation he had injected pure cultures of typhoid bacilli. Legry and Pavone[2] only found fatty degeneration of the livers cells and of the endothelial cells of the capillaries.

In order to throw further light upon the necrosis found in the human liver, we have injected pure cultures of typhoid bacilli into the mesenteric vein of rabbits, this procedure sufficing to carry the bacilli at once into the hepatic circulation, giving them an opportunity to exert their full effect upon the parenchyma of the liver. The cultures were in plain or one per cent. glucose bouillon, and were from twenty-six hours to twenty days old. The culture which was used for these experiments had been obtained from the spleen of a patient who had died of typhoid fever in the Johns Hopkins Hospital, and had been thoroughly tested as regards its growth on the customary media.

The method of procedure was as follows: The animal having been first etherized, a laparotomy was done under strict antiseptic precautions, and an injection of the culture was then made into one of the larger mesenteric veins by means of a sterilized Koch's syringe; the wound was next carefully closed with silk stitches, and celloidin dressing placed over the line of incision. Although we laparotomized thirteen rabbits, in only ten of these was the injection of the culture into the circulation satisfactorily done. In one of the three unsatisfactory cases the hemorrhage following the withdrawal of the needle was of

[1] "Intravenous Inoculation of Rabbits with the Bacillus Coli Communis and the Bacillus Typhi Abdominalis," Johns Hopkins Bulletin, July, 1891, No. 14.

[2] "Nuovi Punti di vista nello studio della quistione del potere patogeno del bacillo del Tifo degli anamali da sperimento." Giornale Internacionale delle Scienze Mediche, 1888.

such amount as to call for the use of a ligature, which was followed by hemorrhagic infarction of the bowel and premature death of the animal. In one case the culture was accidentally injected between the layers of the mesentery, and in one case an attempt was made to inject the culture into one of the smaller branches of the mesenteric artery, which was also followed by hemorrhagic infarction and early death of the animal. We will now give brief protocols of some of our experiments.

Experiment No. 1. May 12, 1891, 10 A.M. Small white rabbit; 0.3 c.cm. of a twenty-six hours' old culture of typhoid bacilli in plain bouillon injected into one of the larger mesenteric veins; May 14th, 10 A.M., forty-eight hours after injection, the rabbit was killed. No peritonitis; stomach contained a large quantity of food; moderate amount of fluid feces in small intestine; no enlargement of Peyer's patches or solitary follicles; spleen small, dark red; kidneys normal; liver shows beneath the capsule, on upper and lower surfaces a few roundish or irregular-shaped whitish nodules, 1 to 2 mm. in diameter; there are also to be seen many very minute yellowish dots beneath the capsule and on the cut surface of the organ. Frozen sections show the larger areas to contain coccidia; small areas, however, do not contain these parasites, but are crowded with many fatty granular cells. Cover-slips from blood, liver, spleen, and kidneys negative; cultures (gelatin plates) from blood, liver, and spleen show numerous colonies of typhoid bacilli; kidney plate negative. Hardened sections of liver stained with methylene blue and eosin show, in addition to the areas containing coccidia, many small foci of cell necrosis. These are situated within the lobules. The liver cells within the area have lost their nuclei; but the beam-work of the cells is as yet unbroken; while in some areas a few polynuclear leucocytes are seen within the capillaries; in others the capillaries, especially toward the centre of the areas, contain large numbers of these leucocytes, some of which have even wandered into the dead cells. No bacilli were found on sections.

Experiment No. 2. May 19, 1891, 10 A.M. Small black rabbit; mesenteric vein injected with 0.2 c.cm. of a four days' old culture in plain bouillon. The animal appeared almost overwhelmed by the toxic effects of the injection and died at the end of twenty-three hours. Autopsy made after death; no peritonitis; stomach almost empty; small intestine contains a small quantity of fluid feces; mucous membrane not injected; no enlargement of Peyer's patches or solitary glands; cæcum and large intestine contained a small quantity of hardened fecal matter; the liver darkly congested and shows characteristic psorosperm nodules; no areas of necrosis apparent to the naked eye, either on the surface or on section; spleen slightly enlarged and dark; kidneys normal; no cover-slips were made; frozen sections of liver show marked fatty infiltration of the periphery of all the lobules; no necrosis discoverable except those containing coccidia; gelatin plates from liver, spleen, and kidneys, each give numerous colonies of typhoid bacilli; hardened sections of the liver do not show necrotic areas. Although Experiment No. 2 was negative, as far as regards the finding of necrotic areas, it is worth mentioning that a culture of the typhoid bacillus was obtained from the blood in the foregoing cases at the end of forty-eight and twenty-three hours respectively.

Experiment No. 3. June 24, 1891, 4 P.M. Medium sized rabbit; mesenteric vein injected with a 0.3 c.cm. of a twenty-six hours' old culture in plain bouillon. Animal killed June 29, 1891, 4 P.M. (at the end of five days). No peritonitis; stomach distended with food; spleen much enlarged, dark red; many small minute whitish necroses to be seen beneath its capsule; kidneys normal; liver contains a few coccidia nodules; no other areas of necrosis to be observed; frozen sections of the liver appeared to be normal in all respects, except the presence of coccidia above noted; small intestine contains a medium amount of fluid, yellowish feces; mucous membrane of ileum uniformly congested and extending up the gut to the distance of 14 inches above the ileo-cæcal valve there are to be seen eleven well-marked ulcerations; the largest ulcer involves an entire Peyer's patch and measures 14 by 21 millimetres; the others are small roundish oval ulcers and involve the solitary follicles; all of them contain a central slough, the necrotic process extending in several down to the peritoneal coat of the bowel; all have overhanging edges and show a distinct margin of congestion; large intestine normal; the mesenteric glands distinctly enlarged, and several show upon the surface, and upon section, small yellowish areas of necrosis; cultures from right ventricle, liver, spleen, mesenteric glands, and kidneys; the plates from liver and spleen show numerous colonies of typhoid bacillus, while those from the blood, mesenteric glands, and kindneys give a few colonies of staphylococcus citreus; hardened sections of the mesenteric glands and of the spleen give characteristic caseous areas of tuberculosis; numerous tubercle bacilli found on stained sections; hardened sections of liver normal; sections of the intestinal ulcers show, microscopically, a central necrosis extending down to and involving the peritoneal coat; this necrosis, in each instance, is densely surrounded by an area of polynuclear leucocytes; outside of these were seen a few epithelioid cells in the submucosa; repeated examinations of stained sections of the ulcers failed to discover any tubercle bacilli whatever. With regard to Experiment No. 3, although the microscopical examination proved that the mesenteric glands were tuberculous, the latter condition was definitely excluded from the process, as seen in the intestinal ulcers. The absence of tubercle bacilli in these, the presence of a central area of necrosis surrounded by crowds of polynuclear leucocytes, the involvement of Peyer's patches and solitary glands in the ulcerative process, would we think, bear out the latter statement.

Experiment No. 6. July 2, 1891, 10 A.M. Medium-sized rabbit 0.2 c.cm. of a twenty-four hours' culture in 1 per cent. sugar bouillon injected into the mesenteric vein. Animal killed July 7th, 5 P.M. (three days, seven hours); stomach distended with food; small and large intestine normal; spleen not enlarged; kidneys normal; no necrotic areas found in the liver either on frozen or hardened sections; cultures from the blood negative; those from the bile, liver, and spleen give numerous colonies of typhoid bacilli.

Experiment No. 7. July 24, 1891, 4 P.M. Large black rabbit; 0.5 c.cm. of a thirty-six hours' culture in 1 per cent. sugar bouillon injected into mesenteric vein; after recovery from the anesthetic the animal showed symptoms of acute toxæmia; remained lying on its side with marked tremor of extremities for more than twelve hours; this passed off during the second day; July 29th, 4 P.M. (five days) animal killed; no peritonitis; stomach contains moderate amount of food; small and

large intestine normal; spleen much enlarged, estimated to be three times its normal size, dark purplish-red, firm; kidneys normal; liver presents within its parenchyma, immediately beneath the walls of the gall-bladder, numerous yellowish, white, roundish necroses; walls of gall-bladder much thickened, bile pale, watery, non-viscid, and contains many minute yellowish floating particles; hanging drop made from bile shows a large number of highly motile rods, corresponding in size to the typhoid bacillus; cover-slips from the various organs negative; gelatin plates from bile, spleen, and liver give numerous typhoid colonies; plates from the blood and kidneys negative; microscopical ex-

FIG. 5.

Rabbit's liver, experimental typhoid fever. A necrosis involving parts of three lobules; animal killed on the seventh day after inoculation into mesenteric vein of a pure culture of typhoid bacilli. Camera lucida drawing, Bausch & Lomb objective 2/3, Leitz ocular No. 4. Reduced 1/5.

amination of hardened sections of liver extending through the wall of the gall-bladder show distinct necrosis of the mucous membrane of the gall-bladder, with loss of substance, and in the parenchyma of the liver proper many small and large necroses involving the liver cells; these areas stained brightly with eosin; within the capillaries of several of the areas there are seen many polynuclear leucocytes; one such necrosis occupies almost an entire lobule.

Experiment No. 8. August 14, 1891, 9.30 A.M. Large white rabbit; mesenteric vein injected with 0.4 c.cm. of a twenty days' old culture in 1 per cent. plain bouillon; August 21, 1891, 9.30 A.M. (seven days) animal killed; no peritonitis; stomach, large and small intestine presented nothing worthy of remark; spleen slightly enlarged, dark red; kidneys normal; liver presents, beneath its capsule and on section, numerous minute, yellowish-white necroses; bile green and viscid; cultures from blood, bile, liver, kidneys, and spleen positive; frozen sections of liver show many necroses, consisting largely of fatty granular liver cells; hardened sections of the liver show necroses, both large and small, resembling in all respects those found in the human liver of typhoid fever.

Experiment No. 11. August 31, 1891, 4 P.M. Large rabbit; injection into mesenteric vein 0.5 c.c. of twenty-four-hours'-old culture in plain bouillon; no symptoms of acute toxæmia. September 4th, 10 A.M. (four days after injection), animal killed. No peritonitis; stomach and intestines normal; spleen not enlarged; kidneys normal; liver, numerous very small, round, yellowish necroses under capsule on the upper and lower surface and on section; bile, green, viscid; cover-slips from the organs negative; gelatin plates from blood, liver, spleen, and kidneys negative; plates from bile contained numerous typhoid colonies; frozen sections from liver showed diffuse fatty degeneration of liver cells and many small necrotic foci; hardened sections gave most characteristic foci of cell death, and there was seen within a branch of the portal vein an extensive thrombus.

Experiment No. 12. August 31, 1891, 5 P.M. Large rabbit; 0.5 c.c. plain bouillon culture, twenty-four hours old, injected into mesenteric vein; killed September 13, 1891 (fourteen days after injection); no peritonitis; spleen dark red and much enlarged; stomach and intestines normal; kidneys very pale on section; striation well marked; liver, numerous minute, sharply defined, yellowish-white areas under capsule and scattered through the liver parenchyma; frozen sections show diffuse, fatty degeneration of liver cells, and well-defined areas of necrosis; frozen sections of kidney show marked fatty degeneration of the epithelium of both the convoluted and straight tubes; cultures from blood and kidney negative; liver, spleen, and bile plates give numerous typhoid colonies; hardened sections of liver show many characteristic areas of cell necrosis.

Of the ten rabbits successfully injected into the mesenteric vein with pure cultures of typhoid bacillus, five gave positive results—that is, following this injection, at intervals varying from two to fourteen days, the animals were killed, and upon examination of the liver there were discovered well-defined, localized areas of cell necrosis.

Taken in connection with the localized lesions found in the liver of typhoid fever in man the changes brought about in the rabbit's liver are particularly instructive; for, although in the former it could not be positively determined just how much the presence of the bacillus coli communis contributed to the production of these necroses, we have obtained, in the latter, lesions in all respects identical with those found in the human liver by the injection of pure cultures of the typhoid bacil-

lus, and where any action of the colon bacillus could be certainly excluded. The inference would appear to be a legitimate one that the typhoid bacillus is quite capable of producing the areas of necrosis which are so marked a feature in the human liver.

As in the human liver, so likewise in the rabbit's liver we find the essential lesion to be, first, a necrosis of the cells proper of the organ in localized areas, varying in size from a few cells up to an entire lobule, followed by the accumulation of polynuclear leucocytes within the capillaries of the area and their subsequent migration into the dead cells.

With regard to the relation of the bacilli to the experimentally-produced areas of necrosis, we have not succeeded in finding any bacilli present in the areas; nor have we found colonies of the bacilli on section of the rabbit's liver. Our experience has in this accorded with other workers in this line. The explanation for the failure to find the bacilli on section is most probably due to the fact that the tissues were subjected to the influence of hardening agents immediately after the animals had been killed. As is well known, Fränkel and Simmonds have shown that, by keeping the spleen and liver of rabbits which have been inoculated with typhoid bacilli for periods varying from twenty-four hours to three or more days, especially at thermostat temperature, the bacilli rapidly multiply, so that, whereas, immediately after the death of the rabbit it was with much difficulty that they could be found, at a later time numerous colonies were to be seen on sections of both liver and spleen. Bearing this in mind, it may be allowable to suppose that many of the colonies of typhoid bacilli found on section in the human liver have developed between the death of the patient and the time of subjecting the tissues to preservative agents. This may account for the fact that so many of the colonies are seen to be surrounded by apparently quite healthy liver cells.

To briefly summarize, then, the results of our study of the lesions found in the human liver following upon an attack of typhoid fever, we believe that we may draw the following conclusions:

1. That the term "lymphoid nodule," by which these localized lesions of the liver in typhoid fever have been heretofore generally designated, is an improper one, since it expresses an erroneous idea as to the origin of these peculiar formations.

2. That these so-called lymphomata are not composed of lymphoid cells, as the term would imply, which have wandered in from the portal vessels, but that, on the contrary, these foci represent well-defined areas of cell-death.

3. That in the earlier stages of the necrosis the nuclei which are found in such abundance in these areas are not the nuclei of lymphoid cells (Orth, Hoffman, Siredey), nor do they arise from the multiplication of the nuclei of the hepatic cells (Wagner, Hoffman), but owe their origin

in small part to the disintegration of the nuclei of the liver cells involved, and in greater part to the presence of polynuclear leucocytes which have wandered into the necrotic area.

4. That at a later stage of the process, when the liver cells have become quite broken down into granular detritus, the numerous small, round, and sometimes spindle-shaped nuclei which are observed along the margins of the area, or seen to have penetrated into its centre, are those derived from the connective tissue stroma of the organ (Wagner, Handford).

5. That thus it is probable that the areas of necrosis are replaced by well-defined areas of localized connective tissue.

6. That necrotic areas, resembling in all respects those found in the human liver in typhoid fever, can be reproduced experimentally in the rabbit's liver by the injection into the mesenteric vein of a pure culture of the typhoid bacillus.

7. That although these areas of necrosis in the liver owe their origin to the action of the typhoid bacillus, it has not been possible to more definitely determine in what way this cell death is brought about—that is to say, whether it is due to the immediate presence of the bacilli within the areas of necrosis, or is caused by the action of the so-called toxalbumins which are assumed to be present in the general circulation. It would seem reasonable to suppose that the latter explanation is the more probable, since it is possible to produce well-defined areas of necrosis in the liver of guinea-pigs by the injection of toxalbumins alone, as has been done by Welch and Flexner[1] in diphtheria, and more recently by Flexner[2] by means of minute injections of ricin and abrin.

In conclusion, we desire to express our warmest thanks to Professor Welch, of the Johns Hopkins University, and Professor Councilman, now of Harvard University, for the valuable suggestions made from time to time during the progress of this investigation, and to state that the success attending the experimental work was largely due to their kind advice and assistance.

[1] Johns Hopkins Hospital Bulletin, 1892, vol. iii. No. 20.

[2] "The Pathologic Changes Caused by Certain So-called Toxalbumins," Medical News, August 4, 1894.

A CLINICAL STUDY OF THREE CASES OF PERSISTENT ABSENCE OF THE GASTRIC SECRETORY FUNCTION.[1]

By D. D. Stewart, M.D.,
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In reading Fenwick's admirable lecture on "Atrophy of the Stomach," published a quarter of a century ago, in which he records the gastric condition observed post-mortem in certain cases of this affection, the diagnosis of which in life could be but conjecture,[2] one cannot but feel a sense of gratitude and admiration toward that pioneer in gastric research, Leube, who has made comparatively easy the recognition of diseased gastric processes in life by the aid of the stomach tube and the application of physiological chemistry to their elucidation. Fenwick's studies dealt with the association of cachectic conditions such as originate carcinoma, as of the breast, and certain profound anæmias, otherwise primary, and atrophy of the gastric tubules, a relation which, as concerns the latter, was first suggested by our own Flint[3] some years previously. Since that time this connection has been recorded by many, among whom, in passing, may be noted Nolen[4], Quincke[5], Brabazon[6], Henry and Osler,[7] Ewald,[8] Lewy,[9] and Kinnicutt.[10] These recognized the association of anæmia and the cachectic states with atrophy of the tubules, but their observations as concerns the stomach were confined to post-mortem findings. The application of the stomach tube to diagnostic purposes has since rendered the recognition of the gastric disorder indicative of atrophy less difficult, and given impetus to the more exact study during life of conditions suggestive of atrophy of the mucosa and allied affections of non-secretory activity. Cases of atrophy of the tubules were now not infrequently reported,[11] its diagnosis in certain of which was not alone supported by post-mortem finding, but on the result of chemical examination of the gastric contents in life. Cases in which a clinical diagnosis of atrophy was thus made were reported by Grundzach,[12] Ewald,[13] Jaroski,[14] Boas,[15] Rosenheim,[16] Litten,[17] Einhorn,[18]

[1] Read before the College of Physicians of Philadelphia, April 3, 1895.

[2] And in which the lament occurs, "Unfortunately we are not able to collect and subject to chemical tests the gastric fluid during life as we can the urine."

[3] American Medical Times, September 15, 1860.

[4] Centralb. f. d. med. Wiss., Bd. xx.

[5] Volkmann's Samml. klin. Vortr., No. 100.

[6] Brit. Med. Journ., July 27, 1889.

[7] Amer. Journ. Med. Sci., April, 1886.

[8] Berl. klin Woch., 1886, No. 32.

[9] Ibid., No. 4, 1889.

[10] Amer. Journ. Med. Sci., October, 1889.

[11] Such as Reigel's: Zeitschrf. f. klin. Med., Bd. xi. u. xii. Grundzach's: Berl. klin. Woch., 1887, No. 30. Wolff and Ewald's: Id., 1887, No. 39. Boas's: Münch. med. Woch., 1887, Nos. 41 u. 42.

[12] Berl. klin. Woch., 1887, No. 30.

[13] Id., 1887, No. 30, 1892, Nos. 26 u. 27.

[14] Wiener med. Woch., 1886, Nos. 49-52.

[15] Münch. med. Woch., 1887, Nos. 41 u. 42.

[16] Berl. klin. Woch., 1888. Nos. 51 u. 52.

[17] Zeitschr. f. klin Med., Bd. xiv.

[18] Medical Record, June 11, 1892.

Meyer,[1] and Pacanowski.[2] Most of the recorded cases were associated with pernicious anæmia, or with the cancerous cachexia, or occurred in the senile. The relationship of atrophy of the gastric tubules with pernicious anæmia, or at least with a simple anæmia of high grade was most commonly remarked, although this condition was also not unusually found as the terminal stage of chronic gastritis, the symptoms of which had been evident prior to the development of those of atrophy of the tubules.

The literature of the subject, although rich and interesting, cannot be now especially noticed further; the foregoing remarks and references cited are merely an illustration of the attention it has already received. The present paper is a study of personally observed cases, the condition of which clinically corresponds minutely with that now recognized as underlying atrophy of the gastric tubules, characterized as they are by practically total secretory loss over a long period. These cases are especially of interest occurring without history of gastritis or of causes to originate the same. With their description I shall briefly discuss the causation of this affection, which apparently is not rare, although not commonly recognized, shall note its effects upon the general health and how these may be suitably influenced by treatment.

At the outset it is of prime importance to clearly comprehend what is understood by the gastric condition clinically regarded as analagous to atrophy of the tubules, so that for a moment I shall digress to consider this point. However originated, by this is meant a state in which, over a long and continuous period, there is practically no secretion of gastric juice—of HCl, of pepsin, or of the rennet ferment. Digestion, save of starches, by aid of saliva ingested with the food, does not occur in the stomach, unless, as was evidently the case in one of my patients, regurgitation of a digestive fluid takes place from the duodenum. With the trial meal withdrawn at a period at which the digestive act should be at its height and free HCl present, and at which appropriate tests should demonstrate absence of undigested albumin, the presence of traces only of syntonin, and the presence of proteoses in abundance, and should also show undoubted presence of pepsin and of the milk-curdling ferment in their active condition, none of these occur. The filtrate, if a filtrate may be obtained from the thick masses of undissolved food, is of a degree of acidity only equalling that of the food taken. Instead of the total acidity being from 40 to 65, representing, with acid salts, saturation of the organic bases and salts of the ingesta with HCl, and a fair amount of the last as uncombined acid, the acidity is in the proximity of 4 or 5.[3] Not only are traces of uncombined HCl absent, but the

[1] Zeitschr. f. klin. Med., Bd. xvi.

[2] Wiener med. Presse, 1888, No. 22.

[3] I find the acidity of a baker's roll, such as constituted the Ewald test breakfast, well macerated with 300 c.cm. of water, averages 4 to 6, calculated, as in estimations with the gastric filtrate,

absence also of bound HCl is shown by the quantity of this acid required to be added to the filtrate or to the unfiltered portion before a reaction to free acid may be obtained.[1] The total absence of pepsin is shown by the fact that a filtrate, fortified by the addition of a proper amount of HCl, has no digestive action after many hours in the thermostat. The absence of the rennet ferment is similarly demonstrated by the inability of an appropriate amount of the filtrate added to a measured portion of milk, to solidify the latter.

Starch digestion is normal should the albuminoids be in such a state of division as to admit of the penetration of the saliva. Gastric motility is usually affected, and often profoundly so. In consequence of which, symptoms referable to the stomach occur, and the various remote symptoms commonly so originated also assume prominence. Except in those cases developing as the final stage of gastritis or accompanying gastric carcinoma, gastric dilatation, save in minor degree, is usually absent until late in the affection, when motility has been long affected.

Doubtless, certain of the cases once regarded as those of total suppression of the secretory functions, indicative of atrophy of the mucosa, the diagnosis of which rested upon the examination of the stomach contents alone, were probably mere examples of pronounced, although curable, gastritis, in which, with absence of free HCl, further search for the presence of pepsin and the milk-curdling ferment, rennin, was not made, or, if undertaken, was improperly pursued. Javorski[2] has especially studied the separation of total secretory inactivity, which, when constantly observed over a long period, indicates atrophy of the tubules, and the more curable chronic inflammatory condition, and has given us a method of differentiation which, understandingly applied, has almost the certainty of mathematical demonstration. He pointed out in this particular a fact little regarded by many workers in the field of applied gastric chemistry, that the mere addition of HCl to a non-free HCl gastric secretion, even though the latter be of decided acidity,

on 100 c.cm., and titrated with decinormal alkali. A more varied meal, such as that of Reichmann's—30 grammes of meal powder, 2 grammes of salt, 1 wheaten roll, and 200 c.cm. of water—yields a higher acidity, equalling, according to Reichmann (Deut. med. Woch., Feb. 14, 1889), about 0.1 per cent. As no intimate admixture of the roll and the water ingested occur in the stomach, and liquids are so rapidly passed into the bowel, a higher degree of acidity may often be expected from the roll breakfast in these cases, when withdrawn an hour or longer after ingestion, without the occurrence of fermentative changes to increase the same. Then usually, through the removal of the fluid simultaneously ingested, and lack of secretion to further assist in the solution of the food, the gastric contents are in such thick masses as to block the tube in process of removal. A filtrate in such cases can only be obtained by the aid of filter-pump pressure or by expression through muslin. The acidity here may be higher. I found the acidity of the filtrate from a roll macerated in 180 c.cm. of water to be 9. The slight alkalinity of the admixed saliva is likely to have a certain, although small, amount of neutralizing effect.

[1] I find by experiment that fully as much is required as in the filtrate from a similar meal, macerated for the same time, outside the stomach.

[2] Verhandlungen des Congresses f. Inn. Med., 1888.

is not sufficient to demonstrate the absence of pepsin, should no digestive activity under proper conditions of temperature and the like be shown by the same, unless the amount of HCl added be sufficient to at least satisfy the organic bases and albuminoids of the foods and leave a small excess apparent as free acid.[1]

A more important circumstance, which also has tended to render nugatory the experiments, as commonly obtained, with the gastric filtrate is the fact that pepsin and rennin, as the researches of Ebstein and Greutzner[2] show, exist in the gastric follicles as a proenzyme, only to be converted into the active ferments chiefly through the agency of the HCl of the gastric secretion. Pepsin formation can be but slight in cases in which secretion of HCl is much impaired, and in those cases in which little or no secretion of HCl occurs, that of pepsin and rennin is entirely in abeyance, although the latter may exist in the gastric tubules in some amount as the inactive proenzymes. Slight or no digestive activity is, therefore, often to be obtained, as I have frequently noted and elsewhere remarked,[3] with the gastric filtrate of a case of pronounced, although curable, gastritis, in which, without atrophy of the tubules, little or no bound HCl exists, even though HCl be afterward freely added in the manner mentioned. If, however, HCl be first administered in quantity more than enough to satisfy the salts and albuminoids of the ingesta, the transformation of the ever-present proenzyme into the active ferment is stimulated; and, furthermore, if the HCl be introduced into the *fasting* stomach the formation of pepsin is still more apparent. It was this principle that suggested to Javorski his admirable method of ascertaining the activity of pepsin and of lab formation, the secretion of the latter of which runs parallel to pepsin, and the formation of which is also stimulated by HCl.

The test is as follows: Into the fasting and cleaned stomach in the morning 200 c.cm. of decinormal HCl are introduced and after a halt hour withdrawn, the patient in the interim quietly sitting. The acidity of this filtrate is then ascertained by the decinormal sodium solution, and its digestive activity may be at once noted by placing a small disk of egg (about 1½ cgm.) into 10 c.cm. or more. This, inspected from time to time, is maintained at the body temperature in the incubator for twenty-four hours. Should the formation of pepsin by this means be expected its amount may be roughly estimated by definite dilutions

[1] This same is not essential in the stomach, when, with totally absent free HCl (to the color reagents), digestion may readily proceed, provided, as cannot be the case in our laboratory experiments, saturation of the albuminoids by the loosely bound acid is maintained by continuous secretion. Notwithstanding HCl may at no stage of the digestive phase in the stomach be apparent as free acid to the color tests, proteolysis will not necessarily be imperfect without it, although it will be delayed, as the acid imbibed by the albuminoids is all that is required for peptinization.

[2] Quoted by Javorski: Pflüger's Arch., B. 8, p. 169.

[3] Therapeutic Gazette, November 1, 1893, p. 749-750.

in a row of flasks with $\frac{N}{20}$ HCl, so that each flask contains a diminishing quantity of the filtrate and an increasing amount of the diluent. The percentage of digestive activity is then noted in each tube at the end of twenty-four hours. By this means information is obtained not only as to the presence or absence of secretions of pepsin, but also as to relative quantity. Thus, as remarks Javorski, definite data may be reached as to the extent of the anatomical changes. This method applied to the study of the gastric condition of many of the cases formerly viewed as those of atrophy of the tubules, especially in those of advanced gastritis, demonstrated a very fair, although latent activity on the part of the secretory function; the stimulating influence of HCl was alone needed for the appearance of the digestive enzymes.

Javorski,[1] whose experience with cases of suspected atrophy of the tubules was largely as an affection secondary to gastritis, whence he proposed the term atrophic gastric catarrh, believed, as a result of the fact just mentioned that many of the cases he, as well as others, had formerly viewed as those of total atrophy of the mucosa, were simply examples of gastritis as related, and that, in these, secretion might after a time have been re-established. Only isolated instances in which this test of Javorski's has been applied in arriving at a diagnosis are on record. Reichmann,[2] who has especially attended to this point, thinks that very few cases exist of actual *total* atrophy. Thus in 107 gastric cases, excluding cancer, noted in a certain time, 10, or 9⅓ per cent., were looked upon to all practical purposes as those of atrophy of the mucosa, although in eight of the ten larger or smaller traces of peptinization occurred in the stomach. He still views these cases as practically examples of this affection, although not of extreme grade.

In all the cases reported in which it seemed probable that more or less complete atrophy of the tubules existed, the secretory function was found to be very seriously affected. After the trial meal a very low acidity existed, about equalling that of the food eaten. There was total and persistent absence of HCl, of pepsin, and lab ferment. This condition was associated with impaired motility, and, in many advanced cases, gastrectasis of a certain degree existed in consequence. The causative factor in many of the cases, as remarked, was a past, severe chronic gastritis, symptoms of which had antedated the trouble. The majority of the reported cases were in middle or advanced life and only two were in the young. The symptoms, save in a few, were those of pronounced gastric disturbances, and, also, often of grave anæmia. In nearly all, gastric symptoms were at some time prominent, and in the majority continued to be a conspicuous feature of the superadded malady. Pain in the epigastrium or in the right hypochondrium or par-

[1] Loc. cit. [2] Deutsch. Med. Woch., February 14, 1889.

oxysmal and periodical vomiting were very commonly noted. The physical character of the vomit, too, has been regarded as peculiar, in that the food is ejected in thick undissolved masses.

In the cases I have had under continuous observation for several years the symptoms directly and remotely connected with the gastric condition have been of varied character.

In the case I first describe, symptoms referable to the stomach were surprisingly slight, and would have been passed over without an examination of the gastric contents to ascertain their true significance had I not been especially engaged in such work. In this case a chronic gouty arthritis had existed for some time, invaliding the patient, and symptoms of decided renal inadequacy, indicating fibrosis of the kidney, were evident. A study of the state of the stomach showed continuous total lack of secretion with profound affection of the motor function: as regards the latter, it being frequently the case that food from the evening meal was present in the fasting stomach in the morning. Treatment directed to the arthritis was without marked effect, until attention was especially paid to the stomach, and particularly to the impaired motility. Then, the arthritis, which had so far crippled the patient that she was confined to the house, was removed, and for more than a year she has been free from symptoms of joint trouble, has free use of the limbs, and the indications of nephritis have passed into the background. She has gained largely in flesh, is apparently in perfect physical condition, despite the absence of gastric secretion continuing as before.

The second case came under observation with symptoms of pronounced anæmia, plus others, akin to the condition described by Da Costa[1] as cardiac asthenia. Gastric symptoms were not pronounced, although more prominent than in Case I. Consent to an examination of the stomach contents could be obtained with difficulty, and the only reliable test for motility, that of Leube's, was impossible of application, because of the patient's unwillingness to become accustomed to the use of the tube. Improvement under remedies directed to the gastric condition was so marked and continuous to the present that little excuse could be urged for frequent examinations of the stomach contents: although such examinations were at first frequently made, and after marked and continuous improvement have been repeated every few weeks or months. This patient, also, for more than a year, has apparently been in perfect health, eating heartily, totally without symptoms of indigestion; presumably because gastric motility, at first impaired, has become normal.

The third case, the only neurotic of the three, is also the only one in which gastric symptoms were steadily in evidence and at all suggestive. In this case, in addition to symptoms referable to the stomach, those of

[1] The American Journal of the Medical Sciences, April, 1894.

the nervous system, such as hysterical attacks and epileptoid seizures, have been prominent, the bearing of the last of which when upon the gastric disorder, whether of *post* or *propter hoc* order, is not easy to determine. It is probable that all have a similar origin in an inherently oversusceptible, unstable, nervous system. This case, less interesting and less instructive than the preceding, is, however, not without some points of interest. Treatment directed to a neurosis has been in the main unsatisfactory, as might be expected: that to the gastric condition consisted chiefly in the use of large doses of diluted HCl. Special treatment by the tube and the employment of intragastric electricity were impossible, because of the repugnance of the patient to their employment.

The following are histories of these cases:

Case I.—Mrs. J. K. M., aged fifty-two years. First came under observation May 30, 1892. The prominent ailment then was chronic arthritis, which incapacitated her for walking, and for which she was referred to me for treatment by Prof. Keen. The knees and several of the interphalangeal finger-joints had been swollen and painful for a year; no history of acute articular rheumatism. The knee-joints were especially swollen on the inner aspects; a distinct creaking occurred about the patellæ on flexing them, due, as Dr. Keen and I thought, to deposits of urates in these situations. Considerable pain was felt on movement, so that patient was practically chair-ridden. Several of the interphalangeal joints of both hands were nodulated. None showed any spindling tendency. Beside steady pains in the joints, and that provoked by exercise, attacks of severer pain were common in the elbows, knees, hip-, and finger-joints, and in the cervical spine. These always corresponded in onset with a sharp diminution in excretion of uric acid, as was shown by a long series of observations on the urine.[1] The pains were always more marked preceding and during damp weather or a rain-storm. The patient still menstruated regularly. Skin and mucous membranes were somewhat pale; no decided evidences of anæmia; no icterus; no wasting. Weight, 145 to 150 pounds. The heart was habitually somewhat overacting, but without indications of enlargement. Second sound accentuated at the apex; pulse-tension always raised; the sphygmogram always showing the elevated, full tidal-wave of increased pressure, with the chief descending wave higher than normal. Arterial thickening absent. The amount of urine passed, measured daily over a long period, averaged 1000 c. cm. The mixed twenty-four-hour specimens, which were alone examined, were pale, of low gravity, low urea, low chlorides, and contained occasionally hyaline casts. Albumin was frequently present (to heat or HNO_3) in small amount. The amount of urine was easily influenced by diuretics: piperazin, potassium citrate, or drinking freely of water, causing a great increase. The

[1] Her normal uric acid output, as ascertained by a long series of examinations, was 0·5 gramme. Coincident with sharp attacks of joint pain the amount invariably fell to or below 0.1 gramme, indicating, as I have elsewhere (Therapeutic Gazette, February, 1894) remarked concerning this feature of the case, impaired solution of the quadriurates in the blood, with a tendency to their deposit, as sodium biurate, about the cartilages and synovial tissues of the joints.

indications beside the gouty arthritis were of incipient granular kidney.

Symptoms of indigestion were uncomplained of. Appetite was poor. Bowels were somewhat constipated. She was first under my care for but two or three weeks, going in the summer to Richfield Springs for the baths and massage. No improvement had resulted when seen in the late autumn of 1892. Treatment was re-begun about January 1st following. It was then remarked that the stools had been of pipe-clay color for some time, although other indications of bile retention were lacking.[1] Special symptoms of indigestion were still absent, save that during the menstrual period feeling of decided weight in the epigastrium after food was common.

Examination of the stomach contents first made January 24, 1893, and repeated at intervals of from two to three days for three weeks, showed on all occasions total secretory inactivity. The gastric contents were withdrawn from one to one and one-half hours after the Ewald trial breakfast. The stomach was always cleansed by lavage preceding these and all succeeding examinations to the present, which were made at weekly intervals for the first year and subsequently at lapses of one to three months. The removed contents were often neutral in reaction, but usually had an acidity measured by $\frac{N}{10}$ NaHO, and calculated on 100 c. cm. of filtrate of from two to five. An acidity of ten was rare. It is of interest to note that on one occasion an acidity of eighteen was obtained, evidently due to the presence of fermentation lactic acid at a time when propulsion was especially effected and food habitually lay over night in the stomach.[2]

Care was taken, as is always my practice, to thoroughly break up with a glass rod the removed contents, and subsequently by agitation to mix them, that a filtrate representative of the mass be obtained. Often, because of the coherence and solidity of the removed contents, a filtrate is impossible in these cases without the use of the filter-pump. Sometimes this fails, and expression through cheese-cloth is necessary to obtain a material for examination. Not infrequently during the first few weeks of examination before active treatment of the gastric condition was initiated, and before full doses of HCl were begun, the contents presented a homogeneous, gruel-like appearance of a decided yellow hue, although, curiously, the stools then were of a light-gray color.[3] The color and solution of the foods were apparently due to regurgitation of bile, and very likely of pancreatic secretion from the duodenum, through a relaxed pylorus, thus admitting of partial pancreatic digestion within the stomach, in the absence to inhibit it, of HCl secretion. On these occa-

[1] This curious absence of bile pigment in the stools persisted for several months. It finally disappeared under the use of the stomach douche.

[2] On this occasion, although the stomach was cleansed by lavage before the roll breakfast, as was always done, a sour, musty odor was apparent in the removed contents, and a decided Uffelmann reaction was obtained from the filtrate. Uffelmann's test, usually applied, never save on one other occasion before or since gave this reaction. Here was an isolated instance through a cause not easy to understand, that a condition existed ripe for development of lactic acid producing bacilli.

[3] The administration of 300 c.cm. of an $\frac{N}{10}$ HCl solution alone, and sometimes with pepsin coincident with the trial meal was tried on several occasions, and siphonage practised at varying intervals after the meal, for the purpose of studying the effects of ingested HCl upon the digestive process. It was noticed that with the use of HCl, instead of the yellow-hued, well-solved contents obtained but the day before, the food presented a water-soaked appearance of a green color, with abundance of pieces of roll yet evident.

sions especially, although, as might be supposed, no syntonin was present, there was no large amount of undigested albumin in the filtrate, and AM_2SO_4, with boiling, after the removal of the unchanged albumin, gave often a decided cloud, due to the presence of proteoses. After removal of undigested albumin, NaCl to saturation, in the cold, often, also, gave a precipitate, not, however, increased by the addition of acetic acid. On these occasions no further digestive activity could be obtained from the untreated filtrate in the thermostat. Even a trace of the milk-curdling ferment had been usually absent. Occasionally its presence in minute amount is shown by semi-solidification of milk in the warm chamber after many hours' contact, a soft curd appearing then at the end of about ten hours, instead of the firm, instantaneous solidification normally to be obtained. Traces of the prolab-ferment are more common, the further addition of $CaCl_2$ solution usually tending to cause a soft curd at an average time of one hour.[1]

Examinations by Javorski's method for pepsin and lab and their proferments have been steadily made to the present at intervals of two to four months. The filtrate of the $\frac{N}{10}$HCl solution, the latter of which had been allowed to remain in the stomach from a half to three-quarters of an hour, had an acidity of usually 0.07 per cent. This was always reinforced with strong NCl, to 0.1, 0.15, 0.2, and 0.3 per cent. in several tubes. No further digestive activity is ever shown at the end of twenty-four hours in the incubator, save that the edges of the disk of white of egg become slightly transparent, a condition which normally should be observed after a few moments' contact. This even is not invariable, and several changes of filtrate similarly reinforced by acid produce no further evidences of digestion. There are always indications of traces of the milk-curdling ferment, and of its proenzyme by Javorski's method.

At first, although a continuous, careful study of the gastric conditions was made, treatment was not directed to the stomach, apart from the almost daily use of the warm douche. During this time the influence of certain drugs on the uratic condition was being studied.[2] At this time food was almost invariably found in the fasting stomach in the morning, after supper early in the evening before. This is the more interesting, as no gastrectasia has ever existed. The propulsive function was, therefore, profoundly affected, and it now seems certain that improvement in gastric motility under treatment directed especially to it, such as the stomach douche, the use of HCl, but especially intragastric electricity, rather than the use of drugs for the gouty condition, was the cause of the recovery from the last. Piperazin alone and the use of sodium salicylates were without very noticeable effect upon the joint swelling or pain. Subsequently piperazin employed with an alkali apparently was more efficient. Later, all swelling disappeared, and pain finally ceased under the continued use of intragastric electricity. As absolutely no drug to influence the rheumatic condition has been administered for a year and a half, and all symptoms referable thereto have continued absent, it is apparent that continued attention to the much-impaired motility has been the chief cause of the cure of the former.

[1] Traces of the prolab-ferment C, labzymogen, were noted in all the cases, as is common in this condition, even with the existence of supposed widespread atrophy.

[2] See Therapeutic Gazette, February, 1894. "The Influence of Piperazin on the Urine and Especially on Uric Acid and Urea Excretion."

After she had been under observation nearly a year, and during which time menstruation was regular, indications of the approach of the menopause were evident. This seemingly has but little effect upon her general condition. Vasomotor disturbances, such as flushing and headache, related to the climacteric, were complained of for only a short time. The menopause is now passed. The menses became at first very irregular in appearance, and now have ceased (for over a year) to appear. Her physical condition is very good. No symptoms of indigestion occur, although she eats heartily of ordinary table food without any care in its selection. Gastric propulsion is noted from time to time by aid of the tube. It remains perfect despite discontinuance of treatment, more than a year ago. Blood-pressure is no longer abnormally raised. The urinary output still tends to be a trifle excessive, averaging lately 1600 c.cm. in several days' consecutive examination. Urea excretion is higher (29 grammes), while chlorine is normal in amount (7 to 8 grammes). Albumin is inconstantly present in traces (to the heat test). Casts were not found in several recent examinations.

Case II.—Mrs. M. B., aged thirty-five years. First came under observation in November, 1892. Had always been in good health until four years previously. In the winter and spring of 1888 she became weak, lost appetite, developed breathlessness on exertion, cardiac palpitation, pallor, and was finally forced to keep abed, because of extreme debility. Attacks of syncope often occurred on attempting to assume the erect posture. Her health improved somewhat, without special treatment in the summer of 1888, the symptoms, however, returning again in the autumn. She is said to have again been much prostrated, of great pallor, to have had breathlessness on exertion and attacks of syncope. These last were often alarming in character, and more than once death was anticipated by her attendants. The feet and ankles were a trifle swollen at these times, and numbness and tingling were noted in the extremities. These attacks of extreme physical prostration, as if from a suddenly induced profound anæmia, each of several months' duration, had recurred each year without assignable cause. She remained weak and debilitated in the intervals, although able to be about and attend, after a fashion, to household duties. When first seen she was practically confined to the bed. She presented the appearance of decided anæmia; greater than was found to exist by a blood examination. Extremities were always cold; cardiac palpitation existed, with freely acting heart and relatively accentuated second sound at the apex. There was no detectable cardiac enlargement, and no bruits. Pulse tension low. Pretibial œdema. Face occasionally noted to swell. Examination of lungs, liver, and spleen negative. Stomach outline normal. Knee-jerk, station, tactile sensibility: normal. Blood examination, repeated on several occasions before treatment was begun: Corpuscles normal in outline; no leucocytosis; 2,300,000 red cells; hæmoglobin, 40 per cent. No apparent cause for the condition evident. The menstrual function has been regular until some time after the appearance of the first seizure. Coincident with one of the attacks of anæmia it ceased, and had been irregular since, the periods occurring at long intervals and lasting a week or more, in which much blood was lost. This menorrhagia was not common and at no time preceded the advent of the anæmic seizures, so no causal relation

could here be traced.[1] There were no indications of latent gastric or intestinal ulceration, nor of hemorrhoids. A prolonged survey of the stools showed no loss of blood by the bowel. Malaria was excluded by repeated blood-examinations for plasmodia, and by absence of fever and of exposure. A careful study of the urine was also negative, as regards causation. Albumin, save in traces to the more refined tests (and then probably of extra-renal origin), was absent, as were casts. Urea excretion was quite low even for body-weight (120 pounds); chlorine output was also somewhat diminished.[2] As anorexia was then marked and but little solid food was taken, the low urinary solids, and especially urea, may be thus explained. I have found it quite invariable in all cases with much deficiency in active gastric juice that the urea output is low for body-weight and nitrogenous intake. Chlorine excretion in such cases is, however, usually normal or somewhat excessive for obvious reasons, unless there be coincident renal insufficiency. Gastric symptoms in this case were in the background. Nothing was remarked as to their presence or past occurrence until inquiry especially directed to the stomach, after search for another source for the condition was resultless, elicited a history of digestive disturbance. It appears she had been in the past subject for years to occasional spells of vomiting, lasting sometimes for weeks. None had, however, occurred for some years until the first anæmic seizure appeared. During this, and most of those following, she often felt weight in the epigastrium after food, had nausea, eructations, and occasionally vomited. These symptoms were not evident during the period in which I first treated her, so that an examination of the gastric contents was not made. Under a concentrated, easily digested diet, and the employment of iron in full doses, she greatly improved. At the expiration of three and one-half months the anæmia had disappeared. A few weeks subsequently, treatment had been discontinued, a seizure similar to the others occurred, with analogous symptoms and blood state. I now insisted on an examination of the stomach contents, consent to which could not at first be obtained. The condition found was practically similar to that in the other cases, save that, unlike as in Case I., no biliary discoloration was even present, and the contents removed were always in thick masses, which tended to block the tube. As in the other cases, the acidity was always low—from two to five. No response to free HCl by the color reagents was manifest until a number of cubic centimetres of $\frac{N}{10}$ HCl were added to 10 c.cm. of the filtrate. The filtrate reinforced by a suitable amount of HCl had no digestive action after many hours in the incubator. Lab ferment was absent, and its proenzyme present only in minute traces. Examinations made at short intervals during the first year, and at periods of two to four months subsequently, the last at the date of this writing, give always similar results. Javorski's method, also used on a number

[1] The climacteric was passed soon after she came under my care. No menstruation has now occurred for two years. The indications are, from this fact and great gain in flesh, that that function has now finally ceased.

[2] The average figures over a period of four days during one of the anæmic spells were: Amount, 1505 c.cm.; sp. gr., 1010¼; urea, 13.71 grammes; chlorine, 5½ grammes; uric acid, 0.6 gramme. Color deficient in pigment.

Eight months later, and after recovery and considerable gain in flesh (thirty pounds), the average figures for four consecutive days were: 1620 c.cm.; sp. gr., 1012; urea, 17.60 grammes chlorine, 7.70 grammes; uric acid, 0.6 gramme.

of occasions, gives result as in Case I. Treatment instituted consisted in the use of very large doses of HCl before meals, and either betol or B. naphthol after. The acid was continued for nearly a year and the naphthol for several months, despite a prompt recovery from the asthenic condition. No return of the trouble has occurred, although two years have elapsed since the last attack. Her weight is now 165 pounds; her nutrition is apparently perfect. No symptoms of indigestion ever occur. She states she can eat any food, however indigestible, without more than slight, if any, discomfort. The gastric secretory condition remains exactly as before with examinations made after the trial meal. Javorski's method, however, now shows very slight pepsin formation, not at first manifested, under the stimulating influence of the HCl. Prolonged contact (fifteen to twenty hours) in the incubator brings about solution of the periphery of the disk of white of egg, indicating that there remains yet a slight, though dormant, activity on the part of a few of the pepsin secreting cells, as is common in all of these cases.

CASE III.—Mrs. S. R. E., aged thirty-two years, first came under observation in the spring of 1891. It was then noted that she was of robust build, large frame, weight 135 pounds. Skin very sallow and of a leaden hue; never pregnant. Gastric symptoms had been present for five years—two years prior to marriage. These were, essentially, anorexia: always feels more comfortable when fasting; distress after all food, however light; often sensation of burning in the epigastrium, with nausea and, not infrequently, vomiting; nausea for years invariable on rising, always with retching. Eats but little meat, fancying that intensifies distress. Often craves indigestible food, relishing it, and has sometimes no discomfort after. Bowels irregular. Tongue usually furred; mouth dry; teeth perfect. Menstruated early; always dysmenorrhœa; no uterine disease. Epileptoid seizures appeared about the date gastric symptoms first noticed; these occurred several times a month for several years, subsequently about once monthly—never at menstrual period. The onset of the seizures is announced by a remote warning (one to two days), consisting of pain in the nucha and occiput, and about the left scapula. An immediate warning is also usual: a sensation as if she were being painlessly dismembered. This is succeeded by sudden and complete loss of consciousness, and a fall if in the erect posture. The seizures often occur when by herself; has been injured about the head and body in falling. No coarse clonic spasms occur. There is often a generalized tonicity, and occasionally twitching of the face and extremities. Tongue is never bitten. Unconsciousness lasts from a few moments to half-hour. Headache follows, but no stupor. Patient is a profound neurotic. There is a questionable history of true ague several years before while living in a malarial region. She often has had an afternoon temperature of 102° to 102½°. Repeated blood examination during the pyrexia shows no plasmodia. Chills or sweating do not occur. A number of blood examinations during the first few months of observation showed the conditions of chlorosis. Red cells, 4,000,000 to 4,500,000 (the latter more usual); hæmoglobin, 40 to 50 per cent.

The employment of the stomach tube, the use of which is always strenuously objected to, often causes a hysteroidal seizure, in which there is generalized twitching of the muscles and considerable prostration. As she would not accustom herself to the tube, it could not be

often used for purposes of diagnosis or for treatment. Examinations were, however, made by it at intervals of a month during the first year, and then to the present four times a year. Either the full meal of Leube and Riegel, or more often the roll breakfast was given. The results were always invariably as in the other cases. A very low acidity, equalling only that of the food taken, was obtained save on one or two occasions when evidences of fermentation were present. The gastric contents were coherent, thick, and blocked the tube, and, physically, usually showed no evidences of change. Undigested albumin always abounded, but no syntonin or proteoses. Lab ferment always absent; its proenzyme present in minute traces. No traces of pepsin. Javorski's method, used in a modified form[1] in this case, however, showed, as in the others, that traces could be obtained under the stimulating influence of HCl.

Following the establishment of the diagnosis of absence of the secretory function, accomplished now so readily by modern methods of research, the question that naturally first engages the attention is as to the underlying anatomico-pathological state: whether there be a mere temporary suppression of secretion from neurotic disturbance, or whether the condition be dependent upon atrophy of the tubules. Succeeding this, the problem as to origin of these cases appears one less easy of solution, with absence of history of other disease *in loco*, such as gastritis, from which immediate cause and effect may be traced. Of still more practical importance than either of the foregoing, yet intimately blended with them, is the question of therapy. Each of these I shall now consider somewhat in detail.

The recognition of total secretory loss is, as remarked, a matter of comparative ease by the adoption of the methods outlined, provided one is sufficiently on the alert and gastric symptoms are evident. In none of my cases did the symptoms regarded by Reichmann[2] as typical occur —vomiting of food in thick masses and recurring pain in the epigastrium and right hypochondrium, the last indicative of pyloric irritation. In but one were gastric symptoms prominent. In Case I., by far the most interesting from both a diagnostic and prognostic standpoint, these were slight and might readily have escaped notice. The most interesting feature concerning this case, as remarked, was the presence of gouty arthritis, with symptoms of incipient chronic nephritis, the former entirely cured, the latter at least yet held in abeyance by attention to the gastric propulsive function, obviating stagnation and fermentation of the ingesta. The occurrence of disorders in the joints in diseases of the stomach is not a new observation. Bouchard some years ago noted nodular swelling of the fingers in cases of dilatation of the stomach, and

[1] A large amount of HCl is administered with a small trial meal, and siphonage and tests made *secundum artem*. The filtrate usually had an acidity of from 18 to 20. Free HCl, approximately, 0.01 per cent. No lab ferment; traces of the proenzyme. At the expiration of twenty-four hours in the incubator slight digestive activity shown.

[2] Deut. med. Woch., February 14, 1889.

others have described chronic articular enlargement and pain in association with absence of free HCl in the gastric juice, removed by attention to the stomach. It is significant that in two other cases encountered by myself (hospital out-patients) very markedly crippled joints existed without decided gastric symptoms, yet with the same condition of the stomach. In one, a male, aged fifty years, several examinations of the stomach contents, after the usual trial meal, gave a total acidity of but 2 to 3 in 100 c.cm., with other conditions, also, practically similar to those described in the cases here reported. In this case there was a typical blending of rheumatoid and rheumatic arthritis; the joints of the fingers, toes, wrists, shoulders, ankles, and knees were affected, with many of the smaller joints typically spindled. He was a trifle cachectic. The urine was somewhat diminished in amount, although without decided diminution in urinary solids or urea. Uric-acid excretion was normal (in a number of days' continuous observations); the chlorides were increased; albumin was absent. The joint affection had existed for thirty years. The case passed from view before it could be further studied or observations made as to the effects of treatment on the absent secretory function. In the other case, the notes of which have been mislaid, there was a chronic articular rheumatism without a history of an acute seizure. These cases indicate that the occurrence of joint symptoms associated with those of secretory failure are not uncommon.[1] Whether there was in these two cases the same obvious causal relation existing in Case I. of my series can only be conjectured. The association is certainly of interest.

It is of importance to distinguish between the cases of absent secretory function and those of chronic gastritis—easily done by the employment of Javorski's method for the recognition of the presence of pepsin and the lab ferment. These are, as before remarked, found in fair quantity in chronic gastritis, unless the changes have advanced to marked atrophy of the mucosa. The frequent occurrence of mucus in the removed contents and in the wash-water, and the presence of shreds of mucous membrane, or, microscopically, of numerous epithelial cells, and, in cancer, of pieces of the growth or of true cancer cell-nests, assist in diagnosis. It is of importance, also, that in chronic gastritis and in cancer the acidity of the filtrate after a trial meal is usually considerably higher than in conditions of decided atrophy, or at least of absent secretory activity, whether the last be due to atrophy of the tubules or to mere neurosal suppression of secretion.

[1] Since this was written I have encountered still another case—one of typical rheumatoid arthritis—in which there also exists total secretory loss. This case, occurring in my service at the Episcopal Hospital, is now under observation. With a total acidity of from 1 to 2, at a time which should represent the height of digestion of the Ewald meal, and absence of all digestive secretion, the gastric motor power is so well preserved that no symptoms of indigestion are now evident. A similar condition is evident after more varied meals.

The differentiation of these cases from latent carcinoma (especially the infiltrating variety) is a most important one. Here, too, unless an accompanying neurotic suppression of secretion mislead, rarely likely, Javorski's method is of the greatest utility. For although the absence of free HCl is usual in carcinoma of the stomach, it rarely occurs that a fair amount of pepsin and rennin may not be obtained from the widely diffused pepsin secreting-cells under the stimulating influence of HCl introduced into the fasting stomach. It seems established that in carcinoma very decided atrophy of the tubules is lacking save in the terminal stage, when the presence of obvious symptoms, if not tumor and dilatation, at least of cachexia and marked wasting, permit of diagnosis. A sign not to be overlooked, if coincident diminution in motility occur from the infiltrating carcinoma, as is common, is the early and persistent presence of marked lactic-acid production, apparently never more than an occasional and fleeting accompaniment of the cases of primary secretory failure.[1] A study of the bacteriology of the carcinomatous stomach, to which attention is now being especially directed, may also be of great utility in diagnosis. Oppler[2] has recently remarked a saprophytic, probably lactic-acid-producing baccillus in cases of gastric carcinoma with absence of free HCl. Others, including, in this country, Turck, and also myself, are studying this question, and it is hoped that great assistance may be ultimately rendered in this direction toward the early diagnosis of latent carcinoma of the stomach. Of great importance in the diagnosis of primary suppression of secretion is the absence of fermentative changes in the removed contents for a considerable time after withdrawal, uncommon in carcinoma or in gastritis. In the very large number of examinations made in all seasons in my cases, with the filtrate remaining on the laboratory table often for several days, it rarely occurred that odor of decomposition developed. This seemed so surprising with absence of HCl secretion that the point was carefully noted. It was remarked only on three occasions in the first case, and twice in that of the third. The trial meal was then withdrawn in from an hour to an hour and a half after its ingestion. Had it lain for a longer time, as over night, the case might readily have been different. Doubtless, rarely, gastric carcinoma is preceded by symptoms of nervous anacidity. In cases such as these the diagnosis must remain in doubt until the development of obvious symptoms of carcinoma. Ewald[3] remarks that even for years in women the case may occasionally superficially resemble a neurosis. It is, he states, striking, how a case such as this may grow suddenly worse coincident with the appearance of an intercurrent febrile disease, or a powerful mental shock. He watched two cases for

[1] Noticed but twice in many examinations in Case I., and never encountered in the others.
[2] Deutsche medicinische Wochenschrift, January 31, 1895.
[3] Berliner klinische Wochenschrift, June 27, 1892.

years, both of which he had regarded as nervous in character, which ran on and in which no complaint, to be intelligently interpreted, of grave abdominal disorder was uttered. In one, in two weeks after an attack of influenza; in another, shortly prior to a great sorrow, decided objective symptoms of carcinoma of the stomach and uterus were apparent.

The influence of treatment, especially on the accompanying atony, the prime cause of the symptoms of the absent secretory function, and the long duration of the case, will permit, if the diagnosis can be made in no other way, of a certain opinion. A duration of two or three years, without cachexia and wasting or appearance of other significant symptoms of cancer, however, these may occur later, would certainly indicate that the carcinoma was rather a sequel to atrophy of the tubules than a causative factor in the same.

The question of anatomico-pathological state underlying these cases of absent secretory function is less easy of determination than their recognition as a primary affection. Whether there be mere neurosal suppression of secretion with tubules still largely intact, or whether the condition be dependent upon an almost total atrophy of the same can rarely be readily answered from clinical evidence affirmatively with absolute positiveness. It is, perhaps, only when a series of these cases has been followed to the post-mortem table that great certainty of opinion may be ventured from mere clinical observation as to others with similar symptoms. This much, however, may be said, that the true condition underlying certain of these cases may after a time be assumed with a very fair degree of certainty. We know from abundant evidence that atrophy of the gastric tubules does occur, and has been found accountable not infrequently for profound anæmias. But symptoms suggestive of the same may appear as a neurosis is also established. Litten[1] has seen cases in which HCl and the ferments were absent for weeks, but finally returned. He, however, regards atrophy as practically proved if this absence continues persistently for months. I am unaware of there being any case on record in which suppression of digestive secretion has been totally constantly absent, as in these of mine, over a period of several years with final re-establishment of the same. Whatever the underlying original condition may have been, whether merely one of reflex inhibition, to consider it now in Cases I. and II., at least, other than actual atrophy of the secreting structure, seems irrational. Here, in Cases I. and II., totally free from neurotic taint, in face of complete restoration to health, the secreting function remains absent as before. The steady continuous absence of traces of HCl and under the most favorable conditions for the appearance of the same, the presence of only the merest traces of pepsin and rennin, in their zymogen or forma-

[1] Berliner klinische Wochenschrift, December 17, 1888.

tive stages, after several years of treatment, especially directed to provoke a return to activity of the secretory structure by a therapy so uniformly successful in cases of similar suppression in simple gastritis, is more than suggestive of absence of gland structure. I dwell especially upon this since both Ewald[1] and Einhorn,[2] who have reported similar cases, are loath to describe them as examples of atrophy of the secretory structure.

Ewald[3] who has proposed for this affection the designation *anadenia ventriculi*, in describing a case clinically corresponding exactly with what is regarded as the same, is curiously reluctant to accept this pathological diagnosis, although he almost regards it as forced upon him, occurring as the case does in one free from evidences of a neurosis, from general disease of the nervous system, and from spinal irritation, and with absence of remissions and exacerbations in the symptoms of atrophy.

His case is of great clinical interest, resembling in certain particulars two of my own, and like them entirely regaining the general health under treatment, without a sign of restoration of gastric secretion. It is that of a male, aged twenty-nine years; under observation at the date of the report two and one-half years. He at first presented symptoms of pronounced dyspepsia, fulness and pressure after meals, flatulency, weariness and heaviness in the extremities, anorexia, and emaciation. He was finally forced to abandon his occupation—that of a chemist. Evidences of a neurosis were absent. Many and continuous examinations of the stomach contents, after trial meals and by Javorski's method, always gave similar result, despite the return of perfect gastric motility, re-establishment of the health, and a gain in weight of forty-two pounds. Einhorn, who has described four carefully studied cases, similar as regards the prolonged absence of gastric juice, proposes as a better term than *anadenia ventriculi* that of *achylia gastrica*, expressing, whatever the pathological state, the actual consequence of the condition. This is much as the term albuminuria has been employed in the past to cover both functional and organic renal disturbances. Einhorn's cases (three of which were males and one a female) were under observation, respectively: one about four years; another a trifle less than two years; the others a few months. Gastric symptoms in all had been of long duration: in one, a ruminant, they had existed only in early youth. In another, vomiting and gastric pain had been present for twenty years; in another of the four for ten years. In none is it noted that there are evidences of a neurosis.[4]

In two of my cases, the non-neurotics, the menopause occurred while

[1] Berliner klinische Wochenschrift, 1892, No. 26.
[2] Medical Record, June 11, 1892.
[3] Berl. klin. Woch., 1892, No. 26.
[4] Excepting in one the presence of rumination.

they were under observation. In both, symptoms for some time antedated the early appearance of cessation of the catamenia. In case second the symptoms indicated that the menopause was precipitated by the recurring spells of profound prostration and anæmia. In both of these cases no symptoms incident to change of life have occurred for a long time. In each the menses have now been absent for over a year. The third case (the neurotic), in which epileptoid seizures occur, still menstruates regularly. This patient, under observation for four years, I had for a long time regarded as one of purely neurosal suppression of secretion. At present I can only say that I am still somewhat in doubt as to the pathological underlying condition, but am inclined to regard it also as atrophy of the secretory structure succeeding continued neurosal suppression of secretion. In this case intragastric electricity, employed in Case I. of the series, so successfully as regards influence on motility, could not be resorted to, because of extreme and unreasonable repugnance on the part of the patient to such manipulative procedures. A study of the motor-function by the tube was impracticable for this reason. What most strongly negatives a mere functional derangement in the other of my cases, and is also in this case of value in differentiation, as it should be in that of Ewald's, and, apparently, also in certain of those of Einhorn's, is the complete re-establishment of the impaired motor-function of the stomach, permitting digestion to proceed in the bowel by aid of pancreatic secretion, and leading finally to disappearance of all symptoms, and practical—at least temporary—restoration to health, in spite of continued unintermittent absence of any gastric secretion. This will probably prove a point of very great value in the separation of cases of neurotic (temporary) suppression of secretion from those due to atrophy of the tubules. It is, however, one nowhere mentioned by writers on this subject. Impairment of the gastric motility, leading to atony, seems undoubtedly the prime cause of the direct symptoms in cases of this sort, for treatment directed to derangement of this function almost invariably leads to their disappearance. Whatever the determining cause of the secretory loss, disturbance of the motor-function, if not similarly originated, is soon apt to suffer indirectly through absence of the stimulating influence of constantly secreted HCl on the musculature, and of the normal abundant digestive fluid to promote solution, digestion, and ready transferrence of the food-mass into the bowel. Both motility and the secretory function alike suffering equally and simultaneously, as seems usual in these cases, suggests a common origin in derangement of the nervous mechanism of the stomach, and were motility permanently impaired with secretion, it would lend favor to Meyer's[1] suggestion as to the origin of the idiopathic

[1] Zeitschrift für klinische Medicin., Bd. xvi.

form in disease of Auerbach's and Meissner's plexus, or the vagus or sympathetic alone. But were cases such as I describe, and the case of Ewald's above mentioned, due to mere functional nerve disturbance, then complete (for over a year in two of my cases) restoration of the markedly impaired motility, so that, despite what is eaten, symptoms of indigestion never occur and the general health remains perfect, secretory return should also have followed. For because of the functions of secretion and motility being intimately connected in innervation through the pneumogastric and sympathetic, it seems unreasonable to suppose that complete restoration of faulty nerve functioning in the one would not naturally lead, and after so long a time, to at least partial return to the normal in the other, especially in view of the long employment of a therapy so successful as a secretory stimulant in cases of functional derangement and in gastritis.

These cases may have been *ab initio*, through causes not now apparent, those of mere functional neurotic suppression of secretion, through long-continued reflected disturbance in gastric innervation impairing jointly both motility and secretion, leading to atony with symptoms referable thereto. A suggestion of Stockton's[1] is worthy of note in this particular. In referring to the origin of just such cases, he asks if we may not have to do with an affection which at the start is functional merely, but in which long-continued inhibition of glands is succeeded by atrophy, so that "what is first an amenable condition terminates in structural change and is permanent." Before I had read this plausible suggestion of Stockton's, and, indeed, long before it was thus uttered, my own thoughts had run in a similar channel through study of these cases. It would seem probable that the cases I report, as with that of Ewald's referred to, and perhaps also those of Einhorn's, were, through causes unknown, dependent primarily upon functional disturbance in innervation, leading to gradual suppression of secretion and to atony of the musculature. Finally, long-continued inhibition has led to atrophy of the secreting glands. As to a cause for the original source of irritation, the merest conjecture is alone possible. The attractive view of Stockton's as to reflected eye-strain, so plausible both because of what diverse symptoms refractive error may originate, and because in certain cases studied by Dr. Jones and himself, such error in high degree existed,[2] cannot be said to be more than suggestive. In the only neurotic of my cases, as narrated a highly emotional woman, who has had for several years epileptoid seizures, and on whom

[1] Medical News, December 15, 1894.

[2] Dr. Stockton states: "Without a single exception in the cases thus investigated [the number of which is not cited], there has been found to exist a definite, and, relatively speaking, uniform ocular defect, viz., unsymmetrical astigmatism of high degree, varying from one to five diopters." There also was noted in all irregular ametropia; myopia in one eye; hyperopia in the other, with a lack of correspondence in the axes of astigmatism.

the use of the tube often induces hysterical seizures, reflexed irritation from eye-strain might be readily operative, but unfortunately for the general application of Stockton's suggestion, refractive error is practically absent.[1]

In the first of my cases, a highly intelligent woman of great force of character, in whom disturbance through ocular defect was not especially anticipated, some degree of strain exists. She had been refracted long before she came under observation, and was wearing glasses which she had regarded as suitable, but which Dr. Gould, who examined her at my request, found were very incorrect. The refractive condition noted is:

R. + sph 0.50 + cyl. 1.75 D. ax. 30°.
L. — cyl. 0.25 D. ax. 75°.

The extra-ocular muscles balanced and visual acuity in each eye was perfect. Dr. Gould remarked in his report that "the splendid physical and mental organization has endured this irritating defect in a wonderful way."

The third case, a woman of less intelligence, who rarely employs her eyes for reading or more than coarse sewing, could be induced with difficulty to be refracted. Symptoms of eye-strain had never been apparent. An examination made by Dr. Gould showed a slight refractive error,[2] regarded even by Dr. Gould as too inconsiderable to cause any serious reflexes. The curiously similar ocular condition in Dr. Stockton's cases so interested me that I recently communicated with Dr. Einhorn concerning his four cases of presumable atrophy of the tubules, that in the preparation of this paper data bearing on this point be presented from as wide a range as possible. Dr. Einhorn reports that one case, a physician, examined by Dr. Knapp, showed no ocular defect of any sort; another, examined by Dr. D'Oench, was found to have a very slight degree of astigmatism, which, according to Dr. D'Oench, amounted to but little. A third patient is cross-eyed. The eyes of the fourth have not yet been examined. From this it will be seen that eye-strain must not be regarded as the source of irritation in all cases, if it can, as is doubtless true, be considered a factor of reflected trouble in others. In this particular, it is interesting to state for those favoring the causative influence of eye-strain in gastric disorder of supposed reflected origin, that Knies, in his recent work on the *Relations of the Eye to General Diseases*, remarks that "While there are manifold relations between diseases of the skin and the eye, this is true only to a

[1] When this patient first came under observation and sources of reflected disturbance were searched for, an eye examination was made by Dr. Schneideman, who reported total absence of astigmatic error and a slight hyperopia (less than one D.) in both eyes.

[2] A less degree of compound hyperoptic astigmatism in the right eye, with some simple hyperopia in the left.

slight extent of diseases of the digestive organs apart from a few very general occurrences." So that, on the whole, any view attributing gastric disorder to reflected eye-strain seems to receive no great support. Concerning weakness in the ocular muscles, which was noted in one of my cases (Case III.) by Dr. Gould, the following may be quoted from Knies, to indicate this is rather the effect than the cause of the gastric condition: "Abnormal digestive processes are very frequent causes of general anæmia and hydræmia with their sequelæ—feeble accommodation, weakness of the external ocular muscles (insufficiency)." That there is little doubt of this is illustrated by a case in point. In discussing this question recently with a distinguished practitioner, he informed me that but a few days before he suddenly noted a defect in vision not previously existing. An eye examination disclosed a weakness in the recti of evident acute onset, the source of which was found to be an attack of indigestion from a dietetic error the day previously. Resort to a purgative and care in diet led to the complete removal of the defect in a very short time.

As regards other sources than the eye from which reflected irritation might arise, suffice it to say here, the matter has received careful attention, but none has been found which could be regarded as adequate. It must, however, be remarked, concerning case third, that the neurotic taint here evident might, of course, render many slight and forgotten elements in her life, which would have been without influence upon the balance of an evenly poised temperament, productive of great influence for evil. The cause of many of these cases is, therefore, all but conjecture, as we are still without definite knowledge of the origin of the idiopathic form of supposed atrophy of the gastric tubules.

The question of therapy in these cases may at first view be thought barren, but in reality it is one from which in certain directions much may be expected, and, therefore, of the greatest interest for consideration. The indications are threefold: 1. To endeavor to re-establish gastric secretion, at least until such a time that it is clearly apparent that efforts in this line will continue futile. 2. To obviate the impaired motor function which, with the resulting atony, leads to stagnation of the ingesta, to fermentative changes, and finally to stretching and atrophy of the muscularis. 3. To administer digestants in those cases in which with fairly perfect motor function the general health still suffers, indicating lack of response to the excessive demands thrown upon the pancreas.

Although it is now established beyond question that the chief function of the stomach is its propulsive, and that gastric digestion, at best, is but imperfect and a preparatory process to final complete peptinization in the bowel through the more active pancreatic secretion, and that, moreover, perfect health is compatible with total suppression of gastric

juice,[1] serious impairment in the secretory function is so apt to be accompanied by impairment of motility, that persistent absence of a digestive fluid must always be regarded with concern. However likely, therefore, the condition may be regarded as one of atrophy of the tubules, in which, naturally, efforts to restore lost parts would be futile, treatment should always be early and persistently directed toward such restoration. This is the more indicated since certain of the most efficient secretory stimulants serve the twofold purpose of improving the motor function if impaired, and of preserving it if intact. The remedies of most utility in this direction are the intragastric employment of electricity, the administration of HCl, and the intelligent use of the stomach douche. Electricity I place easily first. A large and increasing experience convinces me of its superlative value in all affections of the stomach characterized by impairment in the motor and secretory function. Under its use alone in numerous instances I have witnessed the return of free HCl and an active digestive secretion in cases of chronic gastritis, in which the secretory function has been long and persistently impaired. I have seen also coincident perfect restoration of a toneless muscularis.

I speak here of the intragastric application of one electrode. Percutaneous application of both poles, for obvious reasons, experience has shown to be practically valueless to influence either the secretory or the motor function. Faradism is especially of service in these cases both as a motor and a secretory stimulant. As percutaneously applied its influence in the latter direction must be *nil*, and as a motor excitant it can only be exerted indirectly through its action on the superjacent abdominal muscles, little can be expected from any save its intragastric employment. Harm even may result from its percutaneous use where atony exists with non-patent gastric orifices. Here stretching of a weakened muscularis may occur, as was suggested by Ewald.[2]

In but one of the three cases reported could intragastric electricity be employed for reasons elsewhere stated. In this case both galvanism and faradism were long and persistently tried, with a technique of proved utility, but in vain.[3] The effects on the greatly impaired propulsion were, on the contrary, as is usual, extraordinary, and thus, also, upon the train of ill-symptoms so originated. The stomach, which, although not dilated, contained frequently in the morning remnants of food eaten the night before, and rarely was found empty at the normal period after even a light meal, was, under the daily steady intragastric use of electricity, finally stimulated to a condition of even hypermotility, so that

[1] As demonstrated in two of my cases, in the reported case of Ewald's, and in at least one of Einhorn's.

[2] Berliner klin. Woch., 1892, No. 26.

[3] Inability to re-establish secretion by this treatment, with restoration of motility, has tended more than anything else to indicate that in this case hopeless atrophy of the gastric tubules exists.

the employment of faradism had to be discontinued for an interval. After the continuous use of faradism, over a period of a number of weeks, not only was the stomach always found empty in the morning, but often it occurred that no remnants of the roll breakfast were present an hour after its ingestion, and five and one-half hours after a full dinner the wash-water contained only traces of food. In this case lavage with the hot douche was also long practised, and HCl was taken in full doses intermittently for a long time. These, doubtless, were also of utility as motor stimulants, although they were employed chiefly for their effect upon secretion. It is, therefore, perhaps unwise to attribute the benefit accruing, resulting finally in practical restoration to health, to the employment of intragastric electricity alone. The stomach douche and HCl had, however, been first used for some time before resorting to electricity, and improvement which had occurred under them, although apparent, was incomparably less than almost at once was noted from the use of intragastric galvanism and faradism. Electricity and the douche were used in this case only. Cases II. and III. refused to resort to the tube for purposes other than diagnosis. In Case II. this treatment was not insisted upon, as the administration of HCl and an intestinal disinfectant seemed efficient. HCl was continued, in doses of 6cc. to 8 c.c. of the dilute acid in 240 c.c. of water one-half hour before breakfast and dinner, for six months, and either B. naphthol or betol were taken after meals for the space of two months. No drugs have now been used for over a year. She has gained fifty pounds in weight, and is apparently in vigorous health, although gastric secretion continues absent as before. Case III. was regarded as a subject little likely to be permanently benefited by local or, indeed, any treatment, so that the use of lavage and electricity were not urged in her case. Improvement in gastric symptoms was striking under the use of HCl alone. This, first prescribed when she came under observation in 1891, has been voltarily, quite steadily, continued to the present. Curiously, symptoms of distress which had been felt for a long time after even light food, and nausea and vomiting on rising in the morning, are always held in abeyance by a resort to the acid, and disappear on its steady employment. When omitted for a short season these symptoms promptly recur. In this case, unlike the other two, HCl was taken after meals, and in several divided doses of 20 to 30 drops at intervals of fifteen minutes.

As concerns lavage, or, more properly, the stomach douche, although of generally acknowledged utility as a secretory stimulant, in atony of the stomach it has been regarded with less favor. I need here only say that my experience, a not insignificant one, is favorable to its judicious employment as a motor excitor, although it has proved far less useful than electricity. Employed with water as hot as can be comfortably borne, either plain or the first one or two portions that are introduced contain-

ing a weak solution of soda, or of soda and salt, and never distending the stomach in the procedure, most gratifying improvements in my hands have often resulted in atonic conditions of the muscularis. In Case I., so used for a time in the early part of treatment, without coincident employment of HCl or of electricity, some improvement in motility was soon noted. Food ceased to appear in the wash-water in the fasting stomach in the morning, and the stomach became emptied earlier than usual after a meal. But on the discontinuance of the douche, although it had been steadily used for some time, the condition of marked atony recurred, only to be finally dissipated with electricity.

It is of interest to briefly inquire as to the mode of action of HCl in these cases, in two of which it seemed of such distinct utility. In Case II., in which the most permanent benefit resulted from the acid, so that finally it could be discontinued, it was taken, as in Case I., in doses of 4 to 8cc., in approximately decinormal solution strength, two to three times daily, shortly before meals. To obviate the disagreeable taste, it was taken through a small stomach tube by aid of a funnel, the tube being passed by the patient into the first part of the œsophagus.[1] The effect of the administration of HCl fasting has been before dwelt upon—its power to convert the potential proenzymes into the active ferments and to promote secretion of the latter.[2] More than this, it is supposed, and on good evidence,[2] to act as a direct stimulant to its own secretion. Under its employment, in cases in which, through catarrhal disturbances, free HCl has been long absent, it has been found to return. In the cases here studied benefit accruing was obviously not in this direction. It could have resulted only from influencing impaired gastric motor power, since, as a local disinfectant, it did not seem indicated. Leube's experiments led him to believe that increased acidity toward the end of gastric digestion was the main cause of the onward progress of the food-mass into the duodenum, and although the later observations of Fleishner[3] did not support his contention, I am inclined to believe from past observations, and more especially the result in these cases, the benefit in which can scarcely be otherwise explained, that HCl has in certain cases a markedly beneficial influence on gastric propulsion. By many the utility of this acid, in cases in which benefit results from it, is believed to be due chiefly to its antizymotic action. But in cases with absent HCl it is practically impossible to administer with comfort to the patient sufficient, after a large meal, to satisfy the albuminoids of the food, and leave even traces apparent as active free acid. As I have elsewhere[4] stated, it is

[1] This is my usual mode of administering large doses of HCl. Habituation to this use of the tube is easily acquired by the more nervous, in whom its passage into the stomach for the purpose of lavage or siphonage of the gastric contents may be always annoying.

[2] See my paper: "A Consideration of Some Modern Therapeutic Agents in the Treatment of Diseases of the Stomach," Therapeutic Gazette, February, 1893.

[3] Berliner klin. Woch., 1877, No. 7.

[4] Therapeutic Gazette, February, 1893.

unquestionable that "as an antifermentative and antizymotic HCl takes high rank. Putrefactive changes in the stomach originating through stagnation of food with deficient secretion, permitting the development and multiplication of bacteria, with the presence of irritating organic acids and poisonous leucomaines, cannot occur with a small excess of free HCl."

But in these cases evidences of fermentative changes in the stomach were, as a rule, singularly absent after the light trial meal usually employed containing little albuminoids, which alone permitted the full dose (4 c.c. to 8 c.c.) of dilute HCl administered, when tried for the purpose, to appear at the end of an hour as traces of free, uncombined acid. Moreover, in Cases I. and II. the acid was always taken fifteen to twenty minutes before meals, so that a good portion of it had disappeared from the stomach at the time of ingestion of food. In Case III., in which continuous relief was experienced from symptoms of indigestion by it, it is habitually taken after meals.

The reason for its probable inutility as an antiseptic in cases such as these, with complete absence of secretion of this acid, also measurably holds for its utter inutility administered as a digestant, even when pepsin (here also absent) is coincidentally used, and in full doses. Without pepsin in these cases it would naturally be inefficient,[1] since peptinization could not of course advance beyond the syntonin stage. With pepsin it would be useless, because sufficient HCl could not be also given to more than very incompletely satisfy the albuminoids; certainly enough could not be administered to impart and maintain a sufficient degree of acidity to the food-mass to admit of peptinization. The amount of acid a given weight of proteid will combine with before saturation of its molecules occurs, permitting traces to appear in the free state, is remarkable. This fact, which I had for a long time insisted upon, is worthy of consideration by all who still thoughtlessly adhere to the ancient, senseless therapy of a single minute dose of HCl after meals, in conditions in which its deficiency is supposed to exist, expecting results from the same. Attention has recently been redirected to the matter by Chittenden in his Cartwright Lectures on Digestive Proteolysis. Quoting Blum's experiments, Chittenden points out that with a daily consumption of 100 grammes of proteids there would be required no less than 4½ litres of 0.2 per cent. HCl daily, and still none would remain to be apparent as free acid, so essential for continued and complete peptinization.

In a series of experiments which I made[2] in Cases I. and III., and

[1] Unlike in that large class of cases so promptly benefited by acid alone, in which with simple absence of *free* HCl slight secretion of acid still occurs, but not sufficient to convert the scarcely diminished propepsin into the active ferment, readily accomplished by the administration of HCl.

[2] See Therapeutic Gazette, February, 1893.

with numerous others unrecorded in which there was merely absence of *free* HCl, I found that, in an hour to seventy-five minutes after the roll breakfast and 300 c.cm. of $\frac{N}{10}$ HCl solution, only traces of this acid were apparent in the filtrate in the free state—the highest as 0.018 per cent. In Case I., when the acid was thus used, the homogeneous, yellow, gruel-like appearance often noticed in the removed contents after the trial meal, and evidently due to regurgitation of a digestive fluid of biliary and probably pancreatic secretion from the duodenum, had disappeared. Portions of the roll were now present unchanged, presenting merely a water-soaked appearance. Should an assistant to digestion be required in these cases, which I regard as rarely likely, and which contention the conduct of the cases supports—two of which have been for over a year in perfect physical condition without it—it must be looked for in another direction than pepsin and acid. If attention to impairment of motility does not cause disappearance of symptoms, and evidences of derangement of health are still apparent, indicating, perhaps, only temporary inability on the part of the pancreas to furnish a digestive secretion to meet the requirements, full doses of a pancreatic preparation or of an active preparation of papain, must be employed. Pineapple juice, the proteolytic power of which is very great, could also be resorted to, although no form of this, I believe, is now on the market. Meyer regarded the duration of the vicarious digestive function of the bowel as limited, at most from a few months to two or three years. That compensatory hypertrophy of the secreting structure of the pancreas may occur is shown in the case of atrophy of the tubules reported by Henry and Osler, in which death finally resulted with symptoms of pernicious anæmia. That perfect bodily nutrition may be maintained for a long time in these cases is demonstrated by the condition in one of Einhorn's cases under observation for four years, by the case of Ewald's, before mentioned, and by Cases I. and II. of my own. Both of the last now weigh more than ever before, eat with impunity, and have no gastric symptoms.

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REVIEWS.

LEPROSY: IN ITS CLINICAL AND PATHOLOGICAL ASPECTS. By DR. G. ARMAUER HANSEN, Inspector-General of Leprosy in Norway, and CARL LOOFT, formerly Physician to the Lungegaarde Hospital. Translated by NORMAN WALKER, M.D., Assistant Physician for Dermatology, Edinburgh Royal Infirmary. With numerous photographs and colored plates. Pp. 145. Bristol, 1895.

THE name of one of the authors of this book, Armauer Hansen, the discoverer of the bacillus of lepra, is in itself sufficient guarantee that something more than a compilation may be expected, and in this respect we have not been disappointed. Dr. Hansen has for more than twenty years been engaged in studying leprosy in Norway and elsewhere, and the good work that he has accomplished entitles him to be listened to with respect. His words are authoritative. The translator, Dr. Norman Walker, has also done his part well, with the result that we have the subject of leprosy presented in a simple, intelligible manner that commands admiration. We know of no better exposition of leprosy in any language. There are certain points upon which authorities differ, but Hansen is fair in all his statements, and everywhere in the book we recognize the hand of science guiding the author. Hansen's view of the position of the bacilli in the cells is opposed by Unna. The author denies the occurrence of the nodular deposits of the disease on the palms and soles, while, as is well known, Hillis's experience with the disease in Africa shows that these regions are not infrequently invaded. In Norway, at least, the palms and soles are very seldom, if ever, attacked, for Danielssen also with his large experience never encountered the disease here. The author divides the varieties of lepra into the "nodular" and the "maculo-anæsthetic," the latter term being used in preference to "anæsthetic." In certain districts in Norway the nodular form predominates; in others the maculo-anæsthetic does not predominate, but is present almost as frequently as the nodular. The chapter on the pathological anatomy is full of interest. The author is by no means optimistic regarding the good results to be obtained from remedies in the treatment of the disease, stating that as yet we are unable to destroy the bacilli with remedies, either internal or external, so that it only remains to us to prevent infection, which can be attained by isolation of those affected. Hansen thinks that the best measures are those which have been taken in Norway, where the lepers are isolated at their own request, and where the communities can get rid of the disease, if they will, since the sanitary authorities have the power to order the leper to live sufficiently isolated from his family, and, if he cannot or will not assent to this, can compel him to enter an asylum. The doctrines of cleanliness and isolation are constantly preached. The State pays all

of the expenses of the lepers in the asylums, and their families are thus relieved of the lepers who, through the disease, are for the most part unfitted for work. We commend the book heartily to all interested in leprosy, and trust that it may reach the eyes of sanitarians as well as of physicians. Its teachings are sound, and help us greatly in dealing with the disease. L. A. D.

ATLAS OF THE DISEASES OF THE SKIN, IN A SERIES OF ILLUSTRATIONS FROM ORIGINAL DRAWINGS, WITH LETTER-PRESS. By H. RADCLIFFE CROCKER, M.D., F.R.C.P., Physician to the University College Hospital, London. Edinburgh, London, and New York.

NOTICE has already been directed by the JOURNAL to the first three fasciculi, issued some time ago, of this magnificent illustrated work on diseases of the skin. New fasciculi have since been published, and the excellence of the plates and the variety and number of diseases depicted warrant us in making critical mention of the parts since received. Fasciculus IV. contains eleven portraits, dealing with herpes zoster, pemphigus, lichen scrofulosus et pilaris, bromide of potassium eruption, and lepra. They represent for the most part typical cases, several of them unusually severe types of eruption, as the pemphigus and bromide cases, the latter especially being an exuberant form, with vegetating and crusted lesions, indicative of the great cutaneous disturbance which this drug is capable of producing. The plates of lepra are good, but lack color, having pale washed-out tints, which are not characteristic nor peculiar to this disease. The macular, marginate lesions, with a wide border, represented in one case, are instructive from a diagnostic point of view.

Fasciculus V. contains a strongly painted portrait of that dread disease, pemphigus foliaceus, of which but few cases have been met with in this country. Crocker's case is much like that depicted in Hebra's *Atlas*, but shows a much less exfoliative state of the skin. The entire skin of the body is involved. We are pleased to note a fair representation of pityriasis rosea, a disease which is not rare in the United States, and one which is often mistaken by the general practitioner for either syphilis erythematosa or rubella (German measles). It bears not infrequently resemblance to both diseases. The portrait before us lacks the lamellar or furfuraceous scaliness so characteristic of the affection, and does not show sufficiently the difference of color between the patches and the sound skin. Two cases of morphœa are given, which are designated by the author circumscribed scleroderma. The cases depicted represent, we should say, somewhat unusual forms of this disease, if we take the well-known series of Guy's Hospital models of this affection as typical. Ichthyosis hystrix unilateralis, pigmented and unpigmented moles, and tinea tonsurans (four portraits) are also given in this fasciculus. It will thus be noted that each fasciculus of the *Atlas* contains many more portraits than are usually given in a single fasciculus of such atlases.

Fasciculus VI. contains urticaria pigmentosa, eczema vesiculosum, eczema pustulosum, hydroa herpetiforme, psoriasis, xeroderma pigmentosum, melanotic sarcoma, and secondary sarcoma of the skin. The first

of these, urticaria pigmentosa, is a rare disease, seldom depicted in atlases. It portrays the form of the lesions, but does not give the peculiar tints which are usually met with in this disease, indicative of their urticarial nature. It is, however, too much to expect that all the portraits should be of equal excellence in a work of such magnitude as this, especially where it is obvious that the brush of more than one artist is responsible for the pictures. A fair portrait of hydroa herpetiforme, or dermatitis herpetiformis, may also be remarked upon. Both the coloring and the lesions are too uniform, we think, to do justice to this disease as usually met with, but the pictures serve for diagnostic purposes and bring out the herpetic peculiarity of the disease which distinguishes it from other diseases with which it used to be confounded.

We commend, as being admirable in every way, the beautiful portrait of xeroderma pigmentosum, a disease which the distinguished author of the *Atlas* has in his earlier works done much to elucidate. It represents a grave case, and shows well the varied lesions usually met with in an advanced stage of this rare disease.

Fasciculus IX. contains three figures representing hydroa herpetiforme (dermatitis herpetiformis of Duhring). We are unable to see the justification of the use of the term "hydroa" for this disease, the word signifying a "watery" lesion, whereas it is well known that the lesions in this disease may be erythematous as well as vesicular or bullous.

There is a good picture of the rare disease pityriasis rubra, showing the peculiar and abundant "papery" desquamation so characteristic. Next we notice a plate with no less than six colored drawings illustrating the several phases of ichthyosis—all rare varieties of the disease, including circumscribed forms which incline to attack the hands. This fasciculus is very profusely illustrated and contains much that is interesting. The coloring is up to the high standard of preceding parts and with a few exceptions leaves nothing to be desired.

Fasciculus X. contains several unusual forms of eruption after vaccination, designated erythema papulatum, impetigo contagiosa, and a rare manifestation not named. Other plates show elephantiasis and ichthyosis of the leg, both ordinary forms of these diseases; seborrhœic dermatitis; alopecia areata; and tinea versicolor. This fasciculus, like the preceding one, is admirable.

As especially noteworthy in Fasciculus XI., we may refer to the portraits of lymphangiectodes, papilloma with xeroderma pigmentosum, milium in an infant, and comedones of childhood. Unusual forms of tinea circinata are also portrayed, as well as lichen planus acutus and acneiform folliculitis of the buttock.

We cordially recommend the *Atlas* to all interested in cutaneous medicine. It is a good and handsome work. The publishers are Macmillan & Co., London and New York. L. A. D.

INDIGESTION, AN INTRODUCTION TO THE STUDY OF THE DISEASES OF THE STOMACH. By GEORGE HERSCHELL, M.D. London. Second edition. New York: G. P. Putnam's Sons. London: Balliere, Tindall & Cox, 1895.

DIGESTIVE PROTEOLYSIS, BEING THE CARTWRIGHT LECTURES FOR 1894. By R. M. CHITTENDEN, Ph.D., Professor of Physiology and Chemistry in Yale University. Tuttle, Morehouse & Taylor, 1895.

THE man who essays a work dealing with indigestion cuts out for himself a task worthy his steel, however powerful. Though he limit it to the simpler processes of the solution of food, properly called primary digestion, he has before him a problem most complex.

Considerations of depth and scope are invariably embarrassing, but the art of the teacher is measured by the felicity of this adjustment.

In the work of Dr. Herschell we realize the truth of his preface, that it is incomplete. It is, however, especially fitted for students, and from that standpoint we may discuss it.

The author yields at times to the temptation to be dogmatic beyond the legitimate possibilities of scientific statement. This is more noticeable in frequent unqualified statements, which are either not quite true, or but partial truths, than in the advancement of faulty theory.

We regard this as especially unwholesome for the student, who is not in possession of enough data to enable him to cull out from an otherwise useful work its defects.

In the practical expounding of the subject it does not seem to us that the author keeps sufficiently prominent the fact that the digestive manifestation is usually a concomitant or consequent symptom of remote causation. While calling attention to the looseness of statement, which is noticeable throughout the work, it is easy to be hypercritical; to exact from the volume a quality beyond its pretensions. Notwithstanding that possible extenuation the author has a right to be considered seriously, and evidence of lack of care in detail is the most serious obstacle to general acceptance of a product.

The contradictory allusions to the antiseptic quality of bile on pages 20 and 46; the marked insufficiency of support offered to the theory that saliva is necessary to the excitement of gastric secretion, on page 15; the lack of effort to elucidate the very complex facts associated with fat emulsification, made more obscure by statements as to the reaction of chyme, on page 17; and emulsification, on page 20, are illustrations of the stumbling-blocks which are unnecessarily left in the path of the student.

The author's conclusion as to the influence of alcohol indigestion is not to be determined from his discussion. His facts presented on page 34 deal with quantities so far in excess of reasonable amounts, as to bear no satisfactory relation thereto, and his entirely sound deprecation of alcoholic abuse on page 32 is equally far from this point.

The greater portion of the work deals with the practical aspects of stomach disease and diagnosis, and is a compact and systematic exposition of the now accepted methods of investigation of digestive conditions, so far as is possible by local means.

Chapters I. and II. are preliminary generalizing statements as to what constitute digestion and indigestion. Chapters III., IV., and V. are devoted to the detail of inquiry into actual conditions existing, and may be followed with satisfaction as a manual of clinical procedure. Occasionally the author drops into a discussion of differential diagnosis in matters too remote and complex for adequate handling in a work of this size.

Chapters VI. and VII. are devoted respectively to treatment and clinical analysis. It is in respect to treatment that one's position is most liable to attack upon this subject or upon any. Hence we do not desire to discuss the details of treatment proposed for any given condition, but to simply reiterate the statement that the ultimate causation is not sufficiently indicated in this study.

The time has come when medical consultants must be broad diagnosticians and rational therapeutists, and to that end the literature of the future emanating from distinguished teachers must be philosophical.

The illustrative cases in Chapter VII., though offering each a suggestion as to local conditions, are none of them full enough in statement to admit a diagnosis as to what ails the patient, however much may ail his stomach. In spite of these criticisms this volume is of decided value, being easy of reference, clear in arrangement, and very full of sound information, much of it the outcome of the author's large experience. The work is valuable to practitioners as well as to students. The bibliography is very complete and the index satisfactory.

The work of Professor Chittenden is of noticeable quantity.

Being but a brief sketch instead of a treatise, its burden as to completeness is less, as its chance of escaping controversy is greater. The first of three lectures deals with the general nature and production of euzymes, and is instructive, clear, and broad. Most interesting is the author's illustration of the method by which katalytic action may occur, and, though no pretence is made that the exact demonstration is offered, the presentation to the reader of a definite hypothesis as to how it may occur is very acceptable in the especial analogy drawn.

A demonstration of the possibilities of more or less digestion by agents other than organic is prominent in this lecture, and is of importance in calling attention to the essentially chemical nature of digestion. The tendency of the author is to simplify the array of products during digestion, as far as is compatible with emphasis of the fact, that digestive products are in series. In lecture second these series are discussed with especial reference to gastric digestion.

A clear enunciation of the co-ordinate relation of pepsin and acid, with the advancement of the view that the two constitute a unit of activity, though not new, is forcible.

The relative affinity of proteids for acids in various stages, particularly bearing upon the detection of free acid, and the withdrawal of combined acid by elements having greater affinity, is very satisfactorily discussed.

The influence of accumulated peptone and the toxic nature of albumoses are considered finally in this lecture.

Lecture third deals with pepsin proteolysis and its relation to the incomplete products of pepsin change. Particularly interesting is the suggestion found in facts now determined as to the nature and fate of lysatin. The volume closes with a thoroughly fair and cogent discus-

sion of the agency and method by which the soluble proteid food becomes finally fitted for appropriation; the process which should be called secondary digestion. Limiting himself too closely, as we think, to the activities resident in the intestinal wall, the author still deals with the subject clearly. This work, small in volume, is by no means so in dignity.

It serves to impress the importance of physiological chemistry, if such impress be necessary.

It presents an orderly statement of complex facts, and is, moreover, a work of graceful utterance, logical thought, and good English.

H. B. F.

A Manual of Diseases of the Ear for the Use of Students and Practitioners of Medicine. By Albert H. Buck, M.D., Clinical Professor of the Diseases of the Ear, College of Physicians and Surgeons, Columbia College, New York, etc. Second revised edition. New York: Wm. Wood & Co., 1895.

We are told in the preface of this manual that "the present edition differs from that of 1889 in the following respects: A new chapter, entitled 'Analysis of Symptoms,' has been introduced as Chapter III.; some additional text has been added to the chapter on 'Chronic Purulent Inflammation of the Middle Ear;' and the section devoted to a description of operations upon the mastoid process has been entirely rewritten and very much amplified. While there are other portions of the book which I should now write somewhat differently if a favorable opportunity for so doing presented itself, the alterations which I should introduce would be of trifling importance, and certainly would not justify the expense of making entirely new plates for these portions of the work."

The new chapters in this edition, enumerated by the author in his preface, the whole of which we have quoted, are characterized by complete and thorough revision, excepting the chapter on Chronic Purulent Inflammation of the Middle Ear. Here the operation of excision or resection of the carious and necrotic membrana tympani and ossicula is entirely ignored. This unfortunately limits the usefulness of the work, as it fails to give the most important directions possible for the treatment of this vital ear malady, and the warding off of its most dangerous sequelæ, mastoid necrosis, sinus-thrombosis, pyemia, and brain-abscess.

It would be difficult to find an aurist of to-day who would reject this invaluable means of treatment of chronic purulency of the ear, which is, after all, only carrying out an ordinary indication of antiseptic surgery in ear disease. And we are, therefore, greatly surprised to find Dr. Buck so far in the rear in so important a matter. As we have often said before in other places, this method of cure of chronic otorrhœa prevents mastoid disease; but an operation for the relief of mastoid necrosis, while it may temporarily relieve the chronic mastoid disease, will not cure the chronic otorrhœa which has caused it. The otorrhœa continues until the necrotic elements in the middle ear are eliminated.

But so long as the chronic otorrhœa continues the patient is liable either to another mastoiditis or some other vital sequela of chronic purulency in the drum-cavity. This is all the more impressive when an otorrhœa continuing after a mastoid trepanation is finally promptly cured by excision of necrotic tympanic tissues.

In regard to "acute diffuse mastoid osteitis," to which the author devotes nine pages, we must insist upon what is now the accepted teaching of *antiseptic* aural surgery, that *acute* mastoiditis is the result of improper treatment of the primary acute tympanic inflammation. If the latter is not secondarily infected, as it too often is, by a septic, irritant treatment with syringes, swabbings, and insufflations of septic medicaments, or inflations of septic secretions from the nares through the Eustachian catheter and inflation-bag, into the drum-cavity, acute mastoiditis will not ensue.

There is only one treatment of any value in acute otitis media, viz., prompt paracentesis if there is not early spontaneous rupture of the membrana tympani, and then antiseptic drainage with a narrow strip of iodoform-gauze or carbolized gauze, and nothing else. All moppings, syringings, etc., now become septic irritants and *stop* the discharge. Thus pathogenic germs which are successfully escaping from the ear are retained and dammed up in the middle ear, the antrum, and the mastoid cavity, and the patient then suffers from a secondary infective inflammation of the mastoid. Too often this is the result of the engrafting of the staphylococci from the finger of the patient or the physician, or some septic medicament, upon the already inflamed ear.

When the proper treatment of acute otitis media, which can be conveyed in a few words, is fully appreciated and carried out, both by physician and laity, the numerous pages now written on "mastoid disease" will be useless, and the number of cases now reported, with increasing frequency it would seem, will diminish. But no diminution of this important disease will take place until its causation by improper treatment is checked.

This manual, according to its title-page, is prepared for "students and practitioners of medicine," but it fails to tell them some important facts, as we have stated, and is entirely too voluminous in matters of interest only to the specialist to be of value to the general physician—a fault it shares with nearly all treatises and manuals on the ear. If these books are to be useful for "students and practitioners of medicine" they must grow smaller instead of larger. Large ones may be of service to the specialist, but bulky ones can never be convenient for the busy medical student or general physician. This manual under consideration contains nearly 500 *closely leaded* pages; a mass of difficult reading. For this, however, the publisher and not the author is responsible.

C. H. B.

PROGRESS
OF
MEDICAL SCIENCE.

THERAPEUTICS.

UNDER THE CHARGE OF

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ANARCOTINE.

DR. WILLIAM ROBERTS calls attention to this alkaloid of opium which he believes has been neglected. This drug which is generally known as narcotine, having been so named by its discoverer, Derosne, has been renamed by Palmer because it has been shown to be quite devoid of narcotic properties. Smyrna opium contains one-fourth as much of this alkaloid (2 per cent.) as it does of morphine (8 per cent.), while in Bengal opium, which is popularly used in India, it is present in larger quantity (morphine 4, against narcotine 6 per cent.). This drug came into notice in India about fifty years ago when quinine was very scarce and expensive. The important investigations of Palmer show incontestably that it is scarcely inferior—and in some classes of cases is superior—to quinine as an antiperiodic. From the observations of nearly one thousand cases he concludes that in 70 per cent. the fever was arrested at the second paroxysm after the medicine was administered; in 20 per cent. the arrest was equally sure, but not so quick; and in 10 per cent. it did not appear to have any curative effect. When there is an intolerance of quinine it was decidedly more efficacious. Garden, a few years later, believed that it was next in value, as an antiperiodic, to quinine. The doses range from one to three grains, and it is perfectly insoluble and tasteless. The chloride is very soluble and of an intensely bitter taste.—*British Medical Journal*, 1895, No. 1807, p. 405.

THE TREATMENT OF DIABETES.

M. ALBERT ROBIN notes that diabetics have a tendency to the loss of mineral substances by the urine. This loss should be avoided; for instance, by the use of sodium chloride in food, naturally salt foods, as olives. To replace potassium, green vegetables or cauliflower and chicory which are rich in that salt; or sixty to ninety grains of potassium tartrate can be given in wine at

meals, or *sel de seignette* [potassium and sodium tartrate] in a quart of water. To remedy the loss of phosphates, magnesium and calcium, the glycerophosphates of these metals may be employed, or the diet may be regulated. Bouillon is not only a tonic to the stomach but also a perfectly assimilable solution of the inorganic salts and represents the principal salines needed by the organism when undergoing a process of demineralization. The drug treatment comprises three groups: 1. Antipyrin. 2. Arsenic, codeine, alkaline earths, quinine sulphate. 3. Valerian, opium, belladonna, potassium bromide. Quinine, the alkalies, and cod-liver oil may be used with either of the three groups, but as adjuvant medication. Of the first group, antipyrin diminishes the general failure of assimilation, the coefficient of oxidation of albuminous material, sulphur and phosphorus, the effect upon the last two being evidence of its action upon the nervous system. It should be given in from thirty to forty-five grains per day, the latter being the maximum, together with one-half this quantity of sodium bicarbonate. This treatment should not be continued for more than five days because it is likely to give rise to albuminuria, an undesirable complication in diabetes. If the diabetic is already albuminuric this drug is almost contraindicated, although in some cases it may diminish the sugar without increasing the albumin. It should also be limited to the fat diabetics; its action is almost *nil* in pancreatic diabetes. Anorexia, rapid emaciation, and feebleness also constitute contraindications. As adjuvant treatment in this first stage are cod-liver oil in one or two teaspoonful doses *per diem*, preferably before the meal. If it is not well borne, butter can be substituted. During hot weather it should be omitted. The oil is indicated for those who become easily chilled and for the descendants of the tuberculous. The alkalies are used as above—to aid the use of antipyrin. In watching over the intestinal functions for constipation, five drachms of *sel de seignette* in a little tepid water, taken before breakfast, is the purgative of choice. Upon the fourth or fifth day the second stage of treatment is entered upon. Of the drugs used for this stage, quinine sulphate lessens the destruction of the albuminoids and of organs rich in sulphur and phosphorus, diminishes the excretion of carbon dioxide and the absorption of oxygen. Arsenic reduces general oxidation and aids nitrogenous and phosphorous metabolism. The alkalies and alkaline earths diminish the functional activity of the hepatic cells, the general activity of all the tissues, and retard the formation of urea and uric acid. Codeine shares in the properties of the opiates, diminishing the coefficients of the nitrogen-utilization, the glycosuria and the polyuria. These drugs are used as follows: Before luncheon a six-grain capsule of quinine sulphate, to be continued for six days, omitted four, and renewed for six days. Before breakfast and after dinner a capsule containing sodium arsenate, lithium carbonate, codeine, and extract of cinchona made up with *poudre thériacale* [the well-known example of polypharmacy], the last merely to obtain its toleration by the stomach, and to administer a mild opiate [less than 1 per cent. of crude opium]. After this has been employed for five days the third stage is reached, and now then comes into use opium for its action upon nutrition, belladonna for the nervous system, valerian to relieve the polyuria, and potassium bromide which diminishes the elimination of the earthy phosphates. These drugs are used as follows: for eight days belladonna, valerian, cinchona, with

the extract *thébaïque*, are administered with lessened, later increased interval of dose. During this time, preferably at the meals, the patient should drink boiled water to which 8 *per mille* of sodium bicarbonate has been added. Cod-liver oil, which may have been continued during the second stage, is now omitted, but the wine of quinquina is continued during the meal. If opium and belladonna are not well borne, or in case of nervous women, potassium bromide in daily amounts of from thirty to forty-five grains in three doses, may be substituted. If the diabetes is florid, of alimentary origin, and the sugar abundant, the first stage of treatment, followed by a prolonged and a severe *régime* may be sufficient for cure. If the sugar does not disappear, but is only reduced, the diminution of sugar should be made greater, not by a continuation of the antipyrin, but by proceeding to the second stage for about fifteen days. If then the sugar, reduced by the antipyrin tends to increase toward the end of this stage, it is necessary to go to the third stage. If after this there is still sugar it is necessry to recommence the series; but whether it disappears or not, it is necessary to interrupt the drug treatment for a month. During this time the alkaline waters should be continued if the patient passes sufficient urea. If, however, this is diminished, or the complaint is made of feebleness, the alkalies should be abandoned and an iron preparation should be administered. If, after any stage of treatment, the sugar has disappeared, the patient should not consider himself cured, but he should resume, only with the greatest care, the use of starches, then fruits and sugar. Of three hundred cases treated, none of whom passed less than twenty-five drachms of sugar daily, twenty-four were completely cured. Probable cures were obtained in twenty-five cases—that is to say, when the sugar had been absent for several months, it would reappear under the influence of strong mental emotions or breaches of diet. But a short course of treatment usually sufficed to bring the glycosuria to an end. These cases which were markedly and permanently benefited were thirty-three in number, never departing from a certain diet and only returning occasionally for treatment. With seventeen others the sugar never disappeared completely, but there were present less than thirteen drachms, and they increased their body-weight.—*Bulletin Général de Thérapeutique*, 1895, 26 liv. p. 9; 28 liv. p. 49.

Dott. V. Ascoli ed A. Zeri report a single case treated by oxygen. A man of sixty presented the usual symptoms. The daily amount of urine was about three quarts of a specific gravity of 1026; albumin was absent; sugar was present in the proportion of twenty-five *per mille*, and the sediment contained abundant crystals of uric acid. The inhalation of oxygen was continued for three months, about forty-five gallons being used each day. After this time a diet rich in carbohydrates did not cause the appearance of sugar in the urine. The purpose of the oxygen was, as stated by the authors, to bring about the power of consuming the sugar in the organism so that it should not be excreted in the urine.—*Il Policlinico*, 1895, No. 11, p. 337.

The Treatment of Enteric Fever on the Antiseptic Principle.

Surgeon R. H. Quill reports that of thirty-six cases two died, a gratifying experience in India where the mortality is considerably higher than in temperate climates. The particular combination he uses is: Purest carbolic

acid 36 minims, spirit of chloroform 2 ounces, compound tincture of cardamon ½ drachm, syrup of hemidesmus 2 ounces, chloroform water 12 ounces. The dose is one ounce with an equal quantity of iced water every second or third hour immediately after food. No untoward symptoms have arisen. Although in one instance the patient took from first to last over two ounces of pure carbolic acid, yet the urine never became black, nor anything approaching that color; occasionally it became high-colored. In India if typhoid fever is treated expectantly with a well-arranged dietary and efficient nursing one expects a mortality of twenty to thirty per cent. If, in addition to this treatment, the above-described method is employed we will find a reduction of the mortality to one ranging from five to twelve per cent.

In this paper we find a novel method of applying an ice-poultice when there is noted distention of the abdomen or the appearance of blood in the stools. A light tin frame is procured about fifteen inches long by twelve inches broad, and with edges two inches high. A floor is formed by attaching to the sides of the frame a piece of flannel lined on its outside with oiled silk. The trough thus formed, having been partly filled with ice in small lumps, is attached by means of tapes to an ordinary surgical cradle, and suspended over the patient in such a way that although the flannel floor of the trough is in close apposition to the whole of the abdomen there is still no pressure upon it.—*Indian Medical Gazette*, 1895, No. 7, p. 249.

Dr. A. P. Hull has treated thirty-two patients during six months. The first eleven were treated with silver nitrate and quinine given continuously from the beginning to the end of the disease. Of these two died. The remainder were treated with guaiacol, internally and externally, with an occasional one-tenth grain dose of calomel, three or four times a day until slight purgation. The guaiacol was given in from one-half to one and one-half drop doses every two hours night and day, according to the tolerance of the patient. The temperature was controlled by the external application of guaiacol and cold sponging. The former lowers the temperature in about thirty minutes, and is preferable to the cold bath as it is equally efficacious and can be applied when the latter is not applicable. The effects will last from three to four hours, but the amount should be small at first—five to ten drops—and gradually increased, as it is liable to give the patient a chill. The largest dose used at any time was twenty drops. It was applied over the abdomen, which was first washed with soap and water and dried. The drug was then slowly dropped on the parts, carefully rubbed in, and covered with oiled silk. The author concludes that (1) the antiseptic treatment is the rational one; (2) guaiacol is a safe remedy and prevents the toxin-poisoning of the later stages; (3) it will lower the temperature when applied externally; (4) the typhoid patients do better by keeping the bowels acting up to a certain point, rather than checking them, and will derive comfort and benefit from daily douching of the large intestine with warm or cool water.—*The Therapeutic Gazette*, 1895, No. 8, p. 508.

The Treatment of Perityphlitis.

Mr. H. A. Caley includes, under this term, the different varieties of local peritonitis originating in the region of the cæcum, but usually dependent

upon lesions of the vermiform appendix. His classification is as follows: 1, simple, non-suppurating; 2, suppuration resulting in abscess; 3, with general peritonitis; 4, relapsing. The last three classes require surgical measures. On the other hand, a large proportion of cases do well under medical treatment. Absolute rest in the recumbent posture is the first essential, and must be maintained until the subsidence of all symptoms, and the disappearance of all local signs. A liquid and carefully adjusted diet should be adopted. Milk when used should be boiled and diluted with barley water or its equivalent; if given in measured doses and at stated intervals, the danger of over-feeding will be obviated. Beef-tea, soups, meat-juices, or whey may be given in connection with or substituted for the milk. Opium is of use when administered with discrimination and discontinued as soon as the conditions which indicated its employment have ceased. These indications are to allay pain and to prevent peristaltic movements of the intestine lying in the right iliac fossa. Leeches, of which three to six may be applied to the right iliac region, are unquestionably capable of affording marked relief, and are frequently employed with highly beneficial results in the earlier stages of the affection. Fomentations are in general use for the relief of local symptoms, alone or in combination with opium or belladonna. Poultices are less desirable as a rule. The local application of an ice-bag has been employed with satisfactory results, but, on the whole, preference may be given to fomentations as being more generally applicable. When the acute stage is passed, evacuation of the bowels may be most beneficial; enemata are the safest and most generally applicable method. When once the acute symptoms have subsided the cautious use of non-irritant purgatives, as the salines, is proper. Sodium sulphate in from one- to two-drachm doses frequently repeated is usually given. Or castor oil, a teaspoonful every half-hour until a stool is obtained, or calomel in divided doses may be preferred. Antiseptic drugs, as salol, naphtol, to check intestinal fermentation, modifying the virulence of the micro-organisms present in the intestinal tract, may be of service. As preventive measures, attention to the digestion, a daily action of the bowels, massage of the abdomen, associated with suitable exercise, may be cited. Important is the administration of an intestinal antiseptic, the most efficient being salol in milk or in a cachet, in ten-grain doses night and morning.—*The Practitioner*, 1895, No. 326, p. 138.

The Results of Haffkine's Anti-choleraic Inoculations in Cachar.

Mr. Arthur Powell furnishes the statistics of cholera which occurred at the Kalam, Karkuri, and Degubber tea estates since the inoculations were commenced, February 9, 1895. No case of cholera has occurred among those who had undergone the second inoculation. Among the uninoculated inhabitants, who numbered 3276, there were 47 cases of cholera, with 20 deaths; among the inoculated, numbering 2936, 3 cases, with 2 deaths. It is quite likely that one of these patients died from the perforation of a dysenteric ulcer, since she was suffering from sloughing dysentery a week before her death. The period covered by the statistics extends from the above date to

May 28, 1895. The results appear to be favorable, and it is likely that the inoculations will become more frequent.—*Indian Medical Gazette*, 1895, No. 7, p. 253.

Calomel.

Dr. W. Blair Stewart has made a clinical study of the use of this drug in 144 cases, chiefly of gastro-intestinal disorders. He finds that 1 grain of thoroughly triturated calomel is equivalent to 5 to 10 grains of the untriturated drug. If given for its purgative effect or for its action on the liver, $\frac{1}{10}$ of a grain triturated is administered every hour or half-hour until free evacuation occurs, or 1 grain is taken, then, if necessary, ounce doses of *liquor magnesii citratis* can be given until free action of the bowels is obtained. The drug is not a direct diuretic *per se*, but it may act indirectly to a slight extent simply by its general stimulating action upon the emunctories of the entire body, thus favoring secretion, excretion, and the elimination of intestinal ptomaines and systemic poisons, all of which act unfavorably upon the secretory and excretory organs when present. Seventeen cases of diarrhœa, due to the ingestion of improper food, were speedily cured by $\frac{1}{12}$ of a grain of triturated calomel and one-half this quantity of podophyllin every two hours. Nine cases of so-called bilious diarrhœa in adults responded rapidly to ½-grain doses every four hours. Sixty-eight cases in children received a saline cathartic as preliminary treatment, then $\frac{1}{100}$ of a grain each of calomel and powdered ipecacuanha was given every one to four hours with "marvellous" results. Twenty-five cases of obstinate diarrhœa, which had been running some days before consultation, responded rapidly to a combination of $\frac{1}{10}$ of a grain of calomel and 1 grain each of lacto-peptine and zinc sulpho-carbolate given every two to four hours. Four cases of diarrhœa in typhoid fever were markedly benefited by the same combination. In the diarrhœa following excessive use of alcoholic liquors, excellent results were obtained from $\frac{1}{10}$ of a grain of calomel and $\frac{1}{100}$ of a grain of podophyllin every three or four hours. This clinical testimony is advanced to show the efficacy of calomel and its eliminative power as contrasted with the evil routine methods of using astringents and opium in the treatment of diarrhœas.—*Journal of the American Medical Association*, 1895, vol. xxiv. p. 836.

Creosote.

Dr. James K. Crook has made use of this remedy for the past six years. From it he finds two very gratifying results: 1. It possesses undoubted power to relieve the fetor of the expectoration in foul-smelling cases of bronchiectasis and phthisical cavities. 2. In small doses (1 to 2 minims thrice daily) it promotes the appetite and tends to stimulate the powers of digestion. Beyond this he has not found that it modifies in any appreciable manner the ordinary course of phthisis. In several cases where it was relied upon as an expectorant, other remedies of this nature having been withdrawn, the results were of a disappointing character, the only apparent effect being the odor which it imparted to the breath. He concludes that its use may with safety be limited to cases manifestly requiring a pulmonary disinfectant, and to

those accompanied with a feeble appetite, flatulency, pyrosis, and other evidences of fermentative action in the stomach and intestines.—*The Post-Graduate*, 1895, No. 9, p. 340.

The Dangers of Strychnine in Snake-bite.

Surgeon R. K. Elliott reports two instances where death followed the use of strychnine as the so-called Australian antidote (method of Dr. A. Mueller). In both instances the antidote seems to have hastened the death of the patients. In the second case there seemed to be no doubt but that it was one of snake-poisoning. The advocates of this method will have difficulty in claiming that the drug is harmless for those who are really bitten.—*Indian Medical Gazette*, 1895, No. 7, p. 259.

Tuberculin.

Dr. Charles Denison cites three instances in which Koch's tuberculin was used in tubercular affections which were not pulmonary—tubercular cystitis and tubercular hip-joint disease (two cases). From the improvement which has been noted in these cases he concludes that there is ample justification for its use in the positive, although sometimes necessarily more or less transitory, immunizing influence.—*New York Medical Journal*, 1895, No. 870, p. 129.

MEDICINE.

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Monarticular Rheumatism.

L. Heidenhain (*Deutsche med. Wochenschr.*, 1895, No. 31, p. 496) calls attention to cases of arthritis affecting a single joint, or occasionally two or three. This occurs in persons without general symptoms; apparently normal temperature (being policlinic cases temperatures were not taken); clean tongue and good appetite. The articular pain is usually only moderately severe, there is tenderness to pressure and on motion, occasionally œdematous swelling over the joint. Traumatism, gonorrhœa, tuberculosis, and syphilis could be excluded. After giving sodium salicylate the symptoms disappear rapidly and do not return. Occasionally, in protracted cases, limitation of motion follows. Out of fifty-one cases of rheumatism observed in the Greifswald Policlinic in three years, thirty-eight were of the kind described. In thirteen cases the shoulder was affected; the wrist, elbow, and ankle being

affected less frequently; the carpal and tarsal and the small joints still more rarely. The chief element on which the diagnosis was based was the rapid subsidence of symptoms after the administration of salicylic acid. The author thinks that in such cases the origin of many obscure cases of endocarditis may be found, although murmurs were not heard in the cases reported.

Senile Epilepsy and Compression of the Carotid.

Naunyn reports (*Zeitschrift für klin. Med.*, Bd. xxviii. p. 217) some observations bearing on the nature of so-called senile epilepsy. In three men, all above the age of sixty-three, with a history of epileptiform attacks, it was possible to bring on attacks precisely similar by compression of the carotids. Naunyn assumed, therefore, that the spontaneous attacks were due also to cerebral anæmia, which, on account of the weak heart and arterio-sclerosis existing in the patients, was not far to seek. Among other signs of anæmia of the brain, in the spontaneous attacks as far as they were observed, there was always a slow pulse; in one case an attack came on when the patient rose suddenly; in another case after a warm bath. Naunyn made experiments with carotid-compression in persons who had never had epileptiform attacks and who had no signs of brain disease or disturbance of the circulation. In persons not more than thirty years old compression of both carotids caused at most mild temporary dizziness. In two men over fifty, with arterio-sclerosis and slight disturbance of the circulation, but without brain symptoms, compression was followed in a few seconds by loss of consciousness, slow pulse, and mild general convulsions. Soon after these experiments were made compression in a case of senile epilepsy was followed by cessation of breathing, so that the subject was abandoned. So far as made, however, the observations seem to prove that senile epilepsy depends on circulatory disturbances due to disease of the heart or vessels. In two of Naunyn's cases bromides had no effect; in the third it seemed to increase the severity of the symptoms. The observations also show that compression of the carotids is of no value in the diagnosis of thrombosis of the basilar artery or of anomalies in the circle of Willis.

The Immunizing and Curative Effects of Antitoxic Serum from Sheep and Dogs in Typhoid Fever.

Beumer and Peiper have extended their investigations, begun in 1887, in immunity against typhoid fever (*Zeitschrift für klin. Med.*, Bd. xxviii. p. 328). They used 1 per cent. peptone-bouillon as a culture medium, and in it cultivated the most virulent typhoid bacilli obtainable. The cultures were killed by exposure to a temperature of from 55° to 60° C. for one hour. Longer exposure or higher temperature decreased the virulence of the culture very materially. (Early experiments had proved that the toxic substance exists in the bodies of the bacteria.) These sterilized cultures were inoculated into sheep at intervals of from three days to two weeks. The amount injected was increased gradually from 1 c.cm. to 100 c.cm. The animals were then bled to the amount of 500 c.cm., the serum treated with 0.5 per cent. solution of carbolic acid, and preserved in the cold and dark.

The serum of uninoculated sheep was found to be unfavorable for the growth of typhoid bacilli, but the antitoxic serum was much more highly so. The immunizing and curative qualities of the serum were tried on animals with most favorable results. The course of typhoid infection is so much more rapid in animals than in man that the results cannot be compared. From their own experiments, however, and from those of others, the authors think it certain that in the serum of animals treated with typhoid toxin immunizing and curative substance exist. The extent to which these facts can be applied to man must be determined by further experiments. The serum obtained from sheep has no dangerous effects when injected in healthy men.

[The curves given by the authors from cases treated in 1891 do not show that the action of the antitoxin serum is at all marked.]

F. KLEMPERER and LEVY (*Berliner klinische Wochenschrift*, 1895, No. 28) have immunized dogs by injecting increasing doses of bouillon cultures of typhoid bacilli into the peritoneal cavity. Serum obtained from the dogs gave immunity to other animals and prevented death if injected after infection. Patients with typhoid fever treated with the serum had mild cases of fever.

THE CAUSE OF DEATH IN PNEUMONIA.

BOLLINGER (*Münch. med. Wochenschrift*, 1895, No. 32) has endeavored to find a more reasonable explanation for the cause of death in croupous pneumonia than has hitherto been available. In investigating the subject he was struck by the large amount of exudate present in many cases. Thus, in fifty out of one hundred fatal cases of pneumonia there was, on the average, more than one kilo of exudate. In only ten cases was the amount less than 500 grammes, the weight of the exudate in the remaining forty cases lying between these two figures. In one case with red and gray hepatization limited to the right lower lobe, the exudate weighed 1300 grammes. The amount of exudate is not dependent alone on the extent of the local process, but is in inverse ratio to the general nutrition and the amount of blood in the body. So in the cachetic, old, and anæmic persons, hepatization is often incomplete—there is a "soft" pneumonia. Such cases in Bollinger's series often presented complications such as chronic nephritis (twenty-four times), cirrhosis of the liver (eight times), tuberculosis and arterio-sclerosis. Cases with slight exudate were twice as frequent as those with large ones in persons above the age of sixty. The effect of pneumonic consolidation on the amount of blood in the body can be realized by considering that whilst in health the lungs contain 7 to 9 per cent. of all the blood, in a severe case of pneumonia they contain from 30 to 40 per cent. The exudate, therefore, has almost the same effect as rapidly recurring hemorrhages associated with fever and infection, for it is composed almost entirely of blood constituents. Aside from the effects on the heart of the alteration of the blood, there are some other changes of considerable interest. Among them is the emaciation after defervescence; but the most remarkable is leucocytosis. Bollinger's explanation of this is that it represents an attempt at regeneration, and he thinks the enlargement of the spleen depends more on the regeneration of leucocytes than on infection. The conclusion then is that the critical symptoms

of collapse and fatal cardiac insufficiency of pneumonia are essentially due to oligæmia. This leads to insufficient nourishment of the heart, which is at the same time weakened by fever and under unusual stress. Perhaps, too, anæmia of the brain may act injuriously on the heart, through the nervous system. According to what has been said, the author thinks that bleeding has properly been abandoned. In addition to the usual methods of maintaining strength saline injections would seem of value.

Collective Investigation in the Use of Diphtheria-antitoxin in Germany.

The *Deutsche med. Wochenschrift* (1895, No. 32) publishes in tabular form the results of a collective investigation on diphtheria treated with and without antitoxic serum. One of the most interesting features of this is the low death-rate of both classes of cases, in marked contrast to some of the earlier reports based on comparatively small numbers. Thus, out of 562 cases treated with antitoxin in Berlin, 84 died, or 15.1 per cent.; whereas, out of 282 cases treated without serum in the same city, 49 died, or 17.4 per cent. Outside of Berlin, 5271 cases treated with serum gave a death-rate of 9 per cent.; and 4197 cases without serum, 14.4 per cent. The totals are respectively 5833 with serum, and 9.6 per cent. mortality; and 4479 without serum, and 14.7 per cent. mortality. The other details are not of much interest in themselves; a later report is promised.

Changes in the Cerebellum in Tabes Dorsalis.

Jellinek (*Deutsche Zeitschrift für Nervenheilkunde*, Bd. vi. p. 231) has examined the cerebellum in six cases of tabes dorsalis. In all cases there was atrophy of the ganglion cells of the corpus dentatum, the cells being long, spindle-shaped or three-cornered instead of round or oval, as in the normal condition. The bloodvessels of the parts had disappeared to a great extent. The fibres penetrating the corpus dentatum, the finer fibres in the lobules of the hemispheres, and, in some cases, also the radiating fibres, were more or less degenerated.

Basedow's Disease.

Pässler (*Deutsche Zeitschrift für Nervenheilkunde*, Bd. vi. p. 21) has analyzed the histories of 58 patients in the Jena Policlinic who showed symptoms of Basedow's disease. Of these, 74 per cent. were women. Of 51 cases 29 had a neuropathic family history, in most cases on the mother's side. Rheumatism and valvular disease of the heart were each noted five times. In all cases but one there was tachycardia; in 43 palpitation of the heart; in 7 more other symptoms of circulatory disturbance. In 10 cases there was no goitre. Exophthalmus was present in 28 cases. The cardinal symptoms of rapid pulse, palpitation, goitre, exophthalmus, and tremor occurred together in only 35 per cent. of cases, one or more being absent in all the others. There was characteristic tremor in 90 per cent. of cases. Nervous irritability was present in 38 cases; restlessness in 19. Severe headache was complained of by 32 patients, dizziness by 23, insomnia by 21. Besides the 5 cases with

valvular affections, 15 had dilatation of the heart; 24 patients complained of a sensation of warmth; 29 cases had increased perspiration; 5 others local hyperidrosis; a number of cases showed periods of emaciation, alternating with times in which there was rapid increase of weight; 17 cases had polydipsia; 4 of these had also polyuria. In 45 per cent. of cases there were severe attacks of diarrhœa, with or without colic. Vomiting attacks occurred in some cases. The irritable cough, first observed by Charcot, was noted in 10 cases; in nearly all cases the breathing was rapid. This the author ascribes to a mild paresis of the respiratory muscles. In 4 cases there were choreic motions, although "choreic tremor" was not observed; 24 cases showed weakness of the voluntary muscles; temporary loss of strength ("giving way of the legs") was observed in 10. The reflexes were usually normal; in 6 cases they were increased; decreased in only 2. There was ankle-clonus in 2 cases. In 1 case the reflexes were similar to those in spastic pseudo-paralysis. Graefe's symptom was noted only 9 times, and was observed by the author in 1 out of 200 healthy persons examined for the purpose. Stellwag's symptom was present 16 times on both sides; 4 times on one side. Möbius's symptom, insufficiency of convergence, was noted in 9 cases; in 1 there was marked paresis of the internal recti with double vision. The pupil-reflex was usually more active than normal. The eye-grounds were normal. In 11 cases the cervical lymphatics were swollen; in 3 cases the spleen was enlarged; 34 cases were anæmic; 4 cases had albuminuria.

Gangrene of the Leg in Typhoid Fever.

F. de Quervain (*Centralblatt für inn. Med.*, 1895, No. 33) reports the case of a man twenty-five years old, who, in the defervescence of a moderately severe attack of typhoid fever, had a rise of temperature with pain in the right leg. These soon proved to be the early symptoms of thrombosis of the right popliteal artery. A combination of dry and moist gangrene followed. Later there were symptoms of thrombosis of the left femoral vein. The right leg was amputated at the lower part of the femur; recovery followed without accident. The right popliteal artery and vein contained laminated, partly organized thrombi, extending far down into the branches of both vessels. Just below the joint was a deep abscess, containing thick, tenacious, grayish-red pus with a peculiar, sour odor. The pus contained motile bacilli of various sizes, which gave all the cultural characteristics of the typhoid bacillus, not staining by Gram's method. There were no cocci. The author thinks the bacilli set up an arteritis, and that the phlebitis and abscess were secondary.

Second Attack of Diphtheria.

The general opinion that diphtheria not rarely occurs more than once in the same individual has recently been opposed by the physicians of the Children's Hospital in Basel. Stocker, of Lucern (*Correspondenz-Blatt für Schweizer Aerzte*, 1895, No. 15), cites the following to show that exceptional instances to the contrary do occur: A boy of fourteen years had diphtheria eight years before, with extensive exudate, followed by severe nephritis and

paralysis of the velum. Two sisters, at the same time, had similar attacks and died of laryngeal stenosis. In September, 1894, the boy had an attack of diphtheria with exudate on both tonsils, half arches, and the uvula. There was albuminuria (0.15 per cent. albumin) lasting ten days. Two weeks from the onset the velum became paralyzed, and there was paralysis of accommodation in both eyes. Although the specific bacilli were not looked for the diagnosis of the author will no doubt be generally accepted. Less probable is his theory of the origin of the later attack, viz., in the setting free of germs left over from the former attack by repairs to the walls and floor of the house in which the patient lived.

Taches Bleuâties.

In a recent article on the above subject V. Y. Bowditch reviews the theories of the formation of these blue spots and records the result of observations on his own part. In nine cases under his own care he never failed to find the pediculus pubis, this confirming the original statement of Moursou that this parasite was always present.

The spots were found in a variety of diseases, not necessarily of a febrile character; they were seen most numerously in a fatal case of meningitis following an otitis media, and were seen also in typhoid, tuberculosis, gastro-duodenitis, acute bronchitis, intermittent fever, and hysteria.

Most of the theories brought forward have been published before, but a new one, apparently not widely known and held by Revilliod, of Geneva, is certainly unique.

Revilliod believes the spots to be due to the excrement of the pediculus, which he states is an insect of very cleanly habits, and that during the night he leaves his usual seat at the root of the hair and deposits his feces in the adjacent parts of the body! Bowditch is inclined to believe that Moursou's original statement of the relation between the pediculus and the taches bleuâties is undoubtedly correct. [With this accords our experience at the Johns Hopkins Hospital, mentioned by Hewetson.—*Bulletin*, vol. v.]—*Boston City Hospital Reports*, 1895.

Stokes-Adams Disease.

Fuchard states that this syndrome has been previously defined as "permanent slow pulse with attacks of syncope and epileptiform attacks;" he confines the term Stokes-Adams disease to the cases where the above symptoms are due to artero-sclerosis.

After a review of the subject and the recording of two new cases he comes to the following conclusions:

1. That under the name of "permanent slow pulse, with attacks of syncope, and epileptiform or apoplectiform seizures," there has been described a syndrome related to different morbid conditions, and that this erroneous nomenclature leads to grave errors in diagnosis, prognosis, and treatment.

2. That under the name of "Stokes-Adams disease" have been classed all the cases of cardio-bulbar artero-sclerosis, as well as those entirely different cases of permanent slow pulse of nervous origin.

3. That in the first case (Stokes-Adams disease) we find as associated symptoms uræmia, angina pectoris, Ménière's disease, etc., all due to arterosclerosis. In the second case (*i. e.*, slow pulse due to nervous diseases) all these accompaniments are wanting.

4. That the therapeutics of the two classes of cases are entirely different.

5. That "fruste" forms of Stokes-Adams disease exist (as cases without permanent slow pulse, etc.), and terminate frequently by sudden death.

6. That this disease is often associated with other diseases, as renal disease, coronary and myocardial disease, etc.

7. That Stokes-Adams disease is due to an intermittent weakness of the bulb and of the heart, and that the therapeutics should be aimed to attack the ischæmia of these organs.—*Archives Gênérales de Médecine*, September, 1895.

Vasomotor Œdema without Albuminuria.

Tchirkaff, in an interesting article, calls attention to a new syndrome, characterized by great œdema without appreciable organic disease. He has studied six cases between 1886 and 1894. The patients were all men of from twenty-five to sixty years of age, generally of a good constitution, though some of them slightly anæmic.

In the cases presenting anæmia the œdema developed slowly over a period of three to four months: in the plethoric subjects it developed much more rapidly.

The anæmic cases strongly resembled the picture presented by cases of chronic nephritis. In the non-anæmic cases there was a different picture, anasarca coming on rapidly in the space of a week or so, the peritoneal cavity filling with fluid, and in the course of the next two weeks the pleural and pericardial cavities also showing signs of exudate. The face, trunk, and extremities all became swollen at the same time.

At times only the upper extremities were œdematous, but usually the lower were most affected. In some cases the œdema affected mostly the neck, face, chest, and upper arms. Certain patients lost the hair from the eyebrows, beard, moustache, and pubes at the time that the œdema appeared; the hair of the head also became very thin and of a softer texture, and changed its color somewhat. The cases all ended in recovery.

Most careful examinations showed that there was no nephritis in these cases; the urine was repeatedly examined, and albumin was never present, showing that they were not cases of chronic nephritis with temporary absence of albumin; there were never, moreover, any signs of uræmia or kindred symptoms.

The blood was carefully examined in all cases, and no changes were found either in the number of corpuscles or the percentage of hæmoglobin; a large percentage of reduced hæmoglobin was, however, always present.

The heart was negative in all the cases, and there were no frank signs of hepatic cirrhosis.

The majority of the patients either showed signs of or gave a history of a preceding lues.

From the increase in the reduced hæmoglobin, and the fact that there were no signs of deficient oxygenation in the lungs, or venous stasis, Tchir-

kaff concludes that we must be dealing with a stasis in the venules and capillaries probably of vasomotor origin.

As the result of his observations he formulates the following conclusions:

1. There exists a form of disease but little known, characterized by general œdema, with effusion into the serous cavities, without albuminuria; at the same time the heart, lungs, liver, and other organs show no change capable of causing œdema from blood stasis.

2. These œdemas are of a vasomotor origin and can be called "general vasomotor œdemas."

3. They are characterized by, and often accompanied by, dilatation of the right heart and moderate artero-sclerosis.

4. They are always accompanied by changes in the blood, *i. e.*, the hæmoglobin is reduced in excess. Beyond this one occasionally sees trophic trouble and pronounced paralysis of the veins of the skin.

5. In most cases these œdemas are of syphilitic origin, and disappear under treatment by mercury and the iodides. It is possible that the œdema without albuminuria, from which the African soldiers suffer, does not always occur from cold or the formation of ptomaines from indigestion.—*Revue de Médecine*, August, 1895.

SURGERY.

UNDER THE CHARGE OF

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LAMINECTOMY FOR COMPLETE PARAPLEGIA PRODUCED BY ANGULAR CURVATURE OF THE SPINE.

GRAY details the following case (*British Medical Journal*, 1895, No. 1789): Boy, aged twelve years, six or seven months after a fall was observed to be growing crooked. A felt jacket had been worn for a time, but had to be abandoned on account of the discomfort produced by it. In July, 1893, there was complete paraplegia which was said to have developed gradually. Sensation was also lost in the legs. On examination a distinct postero-lateral curvature involving the fourth, fifth, and sixth dorsal vertebra was seen. The reflexes, both superficial and deep, were all present. The knee-jerk was exaggerated as was also ankle-clonus. There were no rectal or bladder symptoms.

As all active disease in the spine had subsided, it was decided to remove the arches of the vertebra involved, which was done April 4, 1894. At the

end of six weeks a plaster-of-Paris jacket was applied, and the boy allowed to move about by the aid of crutches. On the 16th of November he was able to walk without any assistance whatever.

A Case of Double Mastoid Disease with Septic Thrombosis of the Lateral Sinus.

Walker contributes the following (*British Medical Journal*, 1895, No. 1789): A woman, aged twenty-four years, was admitted to the Leeds Infirmary with well-marked symptoms of suppuration in the middle-ear, with extension into the mastoid cells on both sides. It was also evident that there was septic thrombosis of one lateral sinus, but beyond the fact of the falling in of the posterior wall of the right meatus there were no symptoms to indicate on which side the disease was more serious. After shaving the head the swelling on the left side was seen to be more pronounced, and the usual incision was made for opening the corresponding antrum. The antrum was carefully cleaned and inspected, but no perforation of the bone was discovered. The bone was next chiseled away over the lateral sinus, which was found at one point covered with pus. On incising the sinus a quantity of pus and broken-down clot were removed. The sinus was then cleaned out by the cautious use of a Volkmann's spoon until a sudden gush of blood showed that the channel was clear, it was then plugged with Horsley's antiseptic wax. The antrum of the opposite side was then opened and drained. Some days later an abscess developed on the back of the neck. After this the patient steadily improved, the optic neuritis almost disappeared; the hearing which had been *nil* in both ears had so much improved on the right side that loud conversation could be heard. The convergent strabismus had almost gone, but the paresis in the left arm and hand still remained.

A Case of Aural Pyæmia Without Sinus Thrombosis Treated by Ligature of the Internal Jugular Vein and Plugging of the Lateral Sinus.

Deanesly records the following interesting case (*British Medical Journal*, 1895, No. 1789): A child, four years of age, developed double otorrhœa eleven days after an attack of scarlet fever. An abscess appeared over the left mastoid, which was incised. The child had the appearance of being very ill. Forty days after the beginning of the disease the patient had a sudden rigor with much lividity, the temperature rose to 103, and the patient vomited several times. These symptoms continued during the next three days, when the author decided to ligate the internal jugular vein on the left side just above the omohyoid muscle, in two places and divide between. The vein showed no sign of inflammation. The left mastoid antrum was next opened and the sigmoid sinus exposed. The sinus showed no evidence of inflammation, and bled profusely on being incised. The hemorrhage was arrested by pressure from a gauze tent. The child died from exhaustion thirteen days after operation. At the post-mortem examination it was found that thrombosis had not occurred, even after the moderate gauze pressure. At one point, in the left sigmoid sinus near the jugular vein, a soft yellow spot was

noticed which had nearly perforated into the arachnoid space. There were apparently two small pyæmic infarcts in the lungs.

Punctured Fracture of the Skull; Symptoms of Compression for Thirty-four Days; Operation; Recovery.

Tubby reports the following case in the *British Medical Journal*, 1895, No. 1789: The patient, aged forty-three years, was thrown from a horse, alighting on his head. He was able to walk home unaided, and at no time was there giddiness or unconsciousness. He was found to have sustained a lacerated wound over the right frontal eminence. The patient objected to any exploration of the wound, as he felt no serious inconvenience. As the condition did not improve, however, he decided to submit to an operation thirty-four days after the accident. At this time the pupils were widely dilated and reacted but poorly to light; pulse was 58, slow and full. The speech was deliberate and hesitating. The mind was clear and memory good. There was slight blurring of both optic disks. There was no paralysis anywhere. The wound was suppurating. Examination of the wound showed that a portion of the skull seven-eighths by half an inch had been depressed for more than half an inch below the surface. The dura was not wounded. The peculiar features of the case are the serious nature of the injury, although attended with such apparently slight symptoms, the depth to which the fragment of bone had been driven and the rapid relief afforded by its removal. The circulation in the superior longitudinal sinus must have been interfered with, but no ill effects seem to have followed.

The Curability of Cancer when the Lymph-nodes are Involved.

Curtis, in an article under this title in the *Medical Record*, vol. 47, No. 7, reports two cases in which there was distinct carcinomatous glandular involvement in which the patients have apparently been cured by removing all of the parts that appeared diseased. The author does not accept Heidenhain's advice to remove the pectoral fascia in cases of carcinoma of the breast and the entire muscle itself if the disease has penetrated into its fibres at any point. He does not think the few cases so far recorded in which this method has been followed have justified a continuation of the practice. The contribution is a plea for the rational treatment of malignant disease, and while not advocating undue conservatism, any unnecessary sacrifice of tissues is properly objected to.

The Treatment of Inoperable Malignant Tumors with the Toxins of Erysipelas and Bacillus Prodigiosus.

Coley, in a review of this subject in the *Medical Record*, New York, vol. 47, No. 3, summarizes his experience as follows:

Up to May 31, 1894, he had treated with mixed toxins twenty-five cases of inoperable sarcoma, eight of inoperable carcinoma, and three of sarcoma or carcinoma. In the cases of carcinoma he had had marked improvement in a number of cases, but no cures. The cases were all very advanced recurrent

tumors. In sarcoma he stated that there were six cases in which he considered there was a reasonable hope of permanent cure. Six months have passed since then, and none of the six cases have shown any recurrence. Of two other cases, which were merely mentioned among the tabulated cases as improving, one has gone on to entire disappearance of the very large tumor and promises to be a cure, and the other, a six-times-recurrent sarcoma of hand, is in perfect health at present, nearly two years since the beginning of the treatment. Since May 31, 1894, he has treated twenty-four cases of malignant tumors, all inoperable and mostly recurrent; with the mixed toxins. Of these cases, thirteen were sarcoma and eleven carcinoma. In many of these cases of carcinoma the injections had an undoubted retarding influence, and in some of the cases the improvement was extraordinary, but in no case did the tumor entirely disappear. In the sarcoma cases the effect was far more marked; and, although in a number of the cases the disease was so far advanced that there could be no possible hope of recovery, still the powerful controlling influence of the toxins was demonstrated, and had the patient's general condition permitted a prolonged use of the injections, the result might have been otherwise.

In three of the thirteen cases the sarcomas have entirely disappeared; and, although no great length of time has elapsed, the results in his older cases make it improbable that relapse will occur. Of his total of thirty-eight cases of inoperable sarcoma, therefore, nine cases promise to be permanently successful.

Temporary Resection of the Nasal Bones in Operations on the Frontal, Ethmoidal, and Sphenoidal Sinuses.

A new method of attacking the frontal ethmoidal and sphenoidal sinuses for the purpose of evacuating pus or removing new growths is reported by Gussenbauer (*Wien. klin. Woch.*, 1895, No. 21), who describes two successful operations, at least so far as immediate results are concerned, in two cases of malignant growths.

The method employed was devised with the view of giving ample room for the operation, and as far as possible freedom from the disfigurement the scar entailed. The operation was as follows: An incision was made, including the skin and periosteum, through the eyebrow along the supra-orbital margin, passing down the side of the nose close to the inner angle of the eye, and uniting with a similar incision on the other side by a transverse incision over the bridge of the nose at the juncture of the osseous and cartilaginous portions. By the use of a sharp chisel the nasal bones were divided from their cartilage, and the nasal processes of the frontal bones were resected; the resection was then carried upward through the lachrymal bones and the portion of the frontal bone forming the superior part of the orbit; after the division of the ethmoid bone and the attachment of its perpendicular plate from the vomer, the bone with the attached superficial structures were turned back in one flap, laying bare the ethmoidal and frontal sinuses. There was very little bleeding, although the nares had been previously plugged on account of hemorrhage. The patient made a good recovery, the bone, which was replaced and sutured in position, uniting by

first intention. The sinuses were packed with iodoform gauze, with drainage through the nares.

In a second operation the incision was carried down the median line of the nose instead of on either side. The nasal bones were separated from each other, resected, and turned back with the attached flaps on either side. Although there was as much room for operating, the operator prefers the first method, as he believes the scars are less noticeable, and the cosmetic effect is therefore better.

Is the Disinfection of Wounds Possible?

Haenel (*Deut. med. Woch.*, 1895, No. 8; *Cent. für Chir.*, 1895, No. 22) believes that the result of the following researches have proved or tend to prove that it is impossible to disinfect living tissues with chemical substances, and that the best means of producing recovery is to provide free drainage for the secretions of the wound, to keep the wound broad open, and absorb all the osmotic secretions in the dressing.

His experiments were conducted on forty-four guinea-pigs. The wounds entered the muscular tissues and were infected by tampons containing a virulent fluid, having in it both streptococci and staphylococci. The tampons remained in for times varying from four to eighteen hours. The wounds were then washed out and dressed with solutions of either 3 per cent. carbolic acid or 6 per cent. of sodium chloride, and were then dressed with tampons and moist gauze dressings soaked in the respective solutions. Six cases were dressed with aseptic dressings without tampons.

Under conditions as similar as possible no distinction could be made between the course of healing whether carbolic or sodium chloride had been employed in the solutions. If the virulence was marked the animals died of acute infection. If the course was chronic there was no distinction possible either in the intensity of the infection or duration of the recovery.

A Case Presenting Symptoms of Cerebral Tumor in which a Large Area of the Skull was Removed for Relief of Intracranial Pressure.

Clark and Morton report the following case in the *British Medical Journal*, 1895, No. 1789: An iron moulder, aged thirty-eight years, was admitted to the hospital on account of very severe headache and fits which he attributed to injuries of the head received when a boy, and to the shock caused by the death of a sister two years previously. The patient's memory had been gradually deteriorating, and his mental capacity had decreased. There was moderate double-optic neuritis, but no hemorrhages. Potassium iodide, pushed to 40 grains three times a day, and antipyrine for the pain seemed to give considerable improvement for a short time. Although the classical symptoms of cerebral tumor were present, there were no definite localizing ones, hence an operation was undertaken to relieve the symptoms by reduction of the intracranial pressure. A circle of bone with a diameter of two and one-half inches was removed from the region of the parietal eminence and portions of skull immediately below and behind it. The dura bulged forcibly through the opening thus made. A small drainage-tube was

inserted and the flap of scalp sutured. The second night the patient was more comfortable than he had been for a long time. Ten days after the operation the wound was healed and the patient was going about the ward. The headaches had entirely gone and he thought the sight better. The improvement mentioned lasted but a short time as far as the fits were concerned, although he had been free from headache during all of this period.

The Regression of Certain Goitres after Exposure of the Hypertrophied Thyroid Body to Air.

The *New York Medical Journal*, vol. lxi., No. viii., contains the following translation of an article on this subject by Bérard in the *Province Médicale:* A young man had an enormous two-lobed goitre which had existed for years, dyspnœa had been complained of for several months, the eyes were slightly brilliant, but there was no exophthalmos, no palpitation or tachycardia and no tremor, but the voice was goitrous. There was some puffiness of the face, and anemia was marked.

The patient was put under ether and a median longitudinal incision through all the soft parts as far as the capsule of the thyroid was made. The capsule was next incised, and an effort made to enucleate the gland. Every attempt, however, was attended with so much dyspnœa that the effort had to be abandoned. The wound was dressed without closing. At the end of forty-eight hours several clamps were removed, and the dressing which had become saturated with serum was changed. There was no longer any functional inconvenience from the growth. Two months after the operation the circumference of the neck had decreased nine centimetres. The wound had almost completely cicatrized, and the ugly shape of the neck was so very much modified that no trace of the deformity could be seen unless the shirt collar was very much open. The normal tone of the voice had returned, and the patient could walk rapidly and even run without inducing dyspnœa. Bérard claims for this method the following advantages: Absolute harmlessness, complete absence of hemorrhage, preservation of the connection of the thyroid body with the trachea, and, finally, the possibility of effecting without the aid of general anæsthesia simple insensibility of the superficial surface with methyl chloride or with cocaine.

Intestinal Incision and Drainage in Acute Peritonitis.

For the intestinal distention, which is frequently observed in grave cases of general peritonitis, Mixter recommends (*Boston Medical and Surgical Journal*, 1895, No. 9) cœliotomy and incision of the intestine. In these cases the great distention causes paralysis of the intestine which usually leads to a fatal result. In such cases the author believes it is safe to open the coils of intestine at as many points as may be necessary to thoroughly drain them. He recommends that they be drawn out of the wound, held over a basin, incised in from one to four places and thoroughly emptied, after which they should be quickly washed off with hot saline solution, sewed up and returned, and the abdominal incision closed. The author has used this method in nearly twenty cases, some of which recovered, and in those that died the

wounds were found to be tight. From the experience just recorded Mixter feels certain that by means of this operation we may save certain patients that otherwise would surely die. In some cases, particularly in those that have had an abdominal incision on the right side, the author secures permanent drainage by introducing a tube into the most prominent part of the cæcum and retaining it as long as necessary. Through this tube salts and stimulants may be introduced if the stomach is not retentive.

The Treatment of Sprained Ankle.

Gibney contributes to the *New York Medical Journal*, vol. lxi., No. 7, a method of treatment that he says involves no loss of time, requires no crutches, and is not attended with any ultimate impairment of function. The method is as follows: A number of strips of rubber adhesive plaster of twelve inches in length and appropriate width are prepared. For a sprain about the external malleolus the first strip is applied, beginning at the outer border of the foot near the little toe and ending on the inner side of the foot about its middle just under the planter arch. The second strip is applied vertically, and passes from about the junction of the middle with the lower third of the leg, down along the side the tendo-Achillis, over the heel, and terminating at a point just above the internal malleolus, but posterior to it. The remaining strips are applied in the same way, each one overlapping the last about one-half of its width, until the whole external malleolus and side of the foot up to the middle third of the leg are covered. It is well to reinforce just under the malleolus by strips passing criss-cross so as to give additional support to the injured part. Care should be taken not to completely encircle the ankle, which might cause injurious compression. For sprains of the tarsal or mid-tarsal joints or other parts the adhesive plaster is to be applied in the same manner, the idea being to give support to the part.

The Final Results Obtained in the Radical Treatment of Hydrocele by the Injection of Iodine.

Spalinger (*Beiträge für klin. Chir.*, Bd. xiii., Heft S.; *Cent. für Chir.*, 1895, No. 27) in studying the radical cure of hydrocele compares the operative method since the antiseptic era with the results obtained by the injection of iodine.

In the 70 cases treated in the Zurich clinic, 48 were treated by the injection of iodine, 30 were cured, in 5 there were relapses, and 13 were not heard from. The average time in the hospital was fourteen to fifteen days. In 17 operations by incision there were 12 cases cured; no relapses, though 5 cases were not heard from. The average stay in the hospital was from thirty-eight to forty-two days.

The author believes that operation should be employed which is least dangerous, which produces the least functional derangement, and is followed by the most rapid cure. The iodine injection, although there is greater chance of relapse, the author believes is the better operation in ordinary cases; in resistant cases with complications the cutting operation should be employed.

The Question of Stitch Abscesses.

Lauenstein (*Beilage zum Cent. für Chir.*, 1895, No. 27) discussed before the German Surgical Congress the cause of the stitch abscesses observed in patients after aseptic operations. He made 216 observations on different suture and ligature materials in regard to their bacteriological relations. Among these bacteria capable of development in so-called sterilized catgut were found 35 times. In 149 samples, 107 were sterilized by dry heat; but 29 were afterward found to contain bacteria capable of development. The bacillis subtilis occurred most frequently, although the micrococcus tetragenus and the staphylococcus albus were also found.

The author summarizes his observations as follows:

1. Clinical observation shows that there are cases of wound-infection which have their origin in the catgut used in the operation.

2. It cannot be definitely settled in any case that the catgut was the source of infection.

3. But it can be said that the so-called sterilized catgut sold in shops contains bacteria capable of development and growth.

4. So long as this catgut is found to contain bacteria, it cannot be free from the suspicion of being the source of infection in wounds.

In the discussion that followed, Kocher, of Bern, said that since 1888 he has employed silk only, and that in his goitre operations before that time he had only 35 per cent. of absolutely primary healings, while since then he has had 85 per cent. Kocher believes that the suture material should be not only aseptic, but also antiseptic.

The past winter he has placed his silk in a watery solution of arsenious acid, and as a result in 35 cases has had absolute primary union.

The Treatment of Intestinal Invagination.

Rydygier (*Beilage zum Centralbl. für Chir.*, 1895, No. 27) basing his conclusions on the study of 84 new cases from reliable sources, summarizes his study as follows:

In acute intestinal invagination:

1. Operation should be resorted to as early as possible after non-operative methods have been thoroughly tried without success. 2. After a laparotomy disinvagination is of value when it can be accomplished without any great difficulty. If the intestinal wall is suspicious looking at the point of invagination, the peritoneal cavity should be walled off with iodoform gauze, or the particular portion of the intestine should be drawn outside of the abdominal wall. 3. When disinvagination is impossible the resection of the invagination is the least dangerous procedure. 4. The resection of the entire invagination should be performed when the invaginating sheath is so markedly altered that there is danger of perforation. 5. The formation of an anus præternaturalis or an entero-anastomosis have usually no place in these cases; only in the presence of collapse is the formation of an artificial anus permissable.

In chronic invagination internal medication and non-operative methods must be employed, but should be abandoned after a week's trial. Disinvagi-

nation is of little avail, although four successful cases have been reported—one at nine months, two at six months, and one at ten weeks. Resection of the invagination is here also the operative method of choice. Entero-anastomosis should only be undertaken in the presence of adhesions. An artificial anus should not be thought of in the treatment of chronic invagination.

Early Motion in Fractures, Especially of the Lower Extremity.

V. Bardeleben (*Beilage zum Centralbl. für Chir.*, 1895, No. 27) gives the results of his treatment of fractures by the use of bandages, permitting the patient to walk about in all cases in which the condition did not demand an amputation, and in which the extremes of age did not make it dangerous. In the past year he has treated 58 cases: 17 fractures of the thigh, 39 fractures of the leg, and 2 patellar fractures, making a total of 181 fractures in which this method has been employed by him. In no case were the results defective. The advantages of the method, both for the limb and for the general health of the patient, were very great. Joints that were not directly involved could be easily moved after the bandages were removed. Muscular atrophy, delirium, and senile catarrh were never present. The time required for healing was shorter than usual.

OBSTETRICS.

UNDER THE CHARGE OF

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ASSISTED BY

WILLIAM H. WELLS, M.D.,

INSTRUCTOR IN OBSTETRICS IN THE PHILADELPHIA POLYCLINIC.

Three Cases of Sclerema Neonatorum.

In the *Zeitschrift für Geburtshülfe und Gynäkologie*, Band xxii Heft 2, Schmidt reports three cases of this rare fœtal malady from the clinic of Erlangen. In all three careful microscopic study was made of the tissues, and bacteriological investigations were also carried out. In each of the children the characteristic tenseness and swelling of the skin were observed, especially noticeable upon the lower extremities and upon the back, and also present upon the upper extremities. The skin upon the chest was free, while little evidence of disease was present in the face. Each patient had a subnormal temperature which resisted warm baths and artificial heat. The limbs could be moved with difficulty, while the children could not be nourished. Post-mortem examination disclosed imperfect expansion of the lungs

and multiple hemorrhages in the lungs and heart. One of the cases was noticeable for the large amount of adipose tissue which had developed; in this patient œdema was not well marked.

In the blood of all three children bacteria were isolated. The capillary-vessels were crowded with round bacteria, which were also found free in the connective tissue. These germs were also present in the parenchyma of the lungs and in the subcutaneous fibrous tissues. When cultures were made in one case, the bacillus proteus was present, and also the bacillus of liquefaction. It is evident that room exists for further study of the germs present in these cases.

It is probable that this disease may properly be considered an infection. In the cases described it was impossible to recognize the point of entry of the infective germs. There was no sign of inflammation about the umbilicus.

So far as the health of the parents might have influenced the children, two of the children were of the same parentage. The mother had already had seven healthy children, and after the birth of those in question she had two others who manifested no deformity.

Modified Milk.

In the *Boston Medical and Surgical Journal*, 1895, No. 12, vol. 133, Worcester describes work done in Dresden for the purpose of rendering cow's milk as nearly as possible an equivalent for mother's milk. The plan followed is based upon the belief that cow's milk is poor in lactalbumin. White of egg is taken as most closely resembling lactalbumin and being easy to obtain. The white of one egg is added to one pint of cow's milk and one and one-half pints of water. To secure the necessary fat, the milk should contain 9½ per cent. of fat, while sugar is added in the form of 13 drachms of milk-sugar. Under strict precautions for asepsis the white of egg is thoroughly beaten with the milk-sugar, and the pint and a half of water slowly added, stirring constantly. This emulsion is then strained through fine linen into sterilized milk. The whole should be slightly stirred or shaken, and poured into a nursing-bottle. If strict cleanliness has been observed, this mixture may be kept for days or even months.

A New Method of Measuring the Outlet of the Pelvis.

Klien describes in the *Monatsschrift für Geburtshülfe und Gynäkologie*, 1895, Band ii Heft 3, a new method of measuring the dimensions of the pelvis at its outlet which he has successfully employed in the clinic at Munich. The best method of making the measurements hitherto known is that of Breisky: it consists in measuring externally the distance between the tuberosities of the ischia, and also in taking the distance from the junction of the sacrum and coccyx to the lower border of the *ligamentum arcuatum*. While this method is fairly accurate and successful, it does not give all the information desired.

With a view of increasing the accuracy of the measurements Klien has devised an instrument composed of a cross-bar terminating in two disks, which

are placed upon the tuberosities of the ischia. To the middle of this bar is joined what is essentially a pair of compasses, having a graded scale fastened across them. With the patient lying upon her back the two disks are pressed firmly upon the skin opposite the tuberosities of the ischia, while the attached calipers or compasses are used to outline a rhomboidal shape, extending from the subpubic ligament to the point of the coccyx. The result is a rhomboidal outline, the various dimensions of which can be computed by the formulæ applicable to a rhomboidal figure.

Klien utilized this instrument to advantage in measuring the pelvis of a woman who had fallen in childhood, breaking her leg and dislocating her hip. As the patient grew to womanhood, the result of this injury was an obliquely contracted pelvis. It was possible by Klien's method to measure accurately the outlet of this pelvis in the manner described.

The Bacteriology of the Birth-canal.

In the *Centralblatt für Gynäkologie*, 1895, No. 38, Stroganoff describes a series of investigations carried on in the clinic of Ott at St. Petersburg. By a method of aspiration he was enabled to obtain the secretions of the cervix uteri without contamination. Cultures were then made from these secretions and also from those of the vagina. His conclusions are that in the pregnant or non-pregnant the cervical canal is, as a rule, sterile. The region of the external os marks the boundary between the presence or absence of bacteria. In all his cases examined it was observed that the mucous secretion of the cervix destroys the life of micro-organisms.

Contracted Pelvis; Delivery at Term of Living Child by the Employment of the Obstetric Lever.

Wassilieff (*Archives de Tocologie*, 1895, No. 9) reports the case of a patient aged thirty-eight years, pregnant for the third time, and having a contracted pelvis whose antero-posterior diameter was between 8½ and 9 cms. As labor proceeded, rotation of the fœtal head failed, and the physcian in attendance attempted to apply the forceps; he could, however, insert but one blade, but rotation was so much improved by the introduction of but a portion of the instrument that the blade was allowed to remain, the turning of the head being facilitated by occasional pressure with the lever. The writer calls attention to the assistance derived in this way, and urges the use of the forceps-blade in the manner described.

Tubal Gestation of Two Months Followed by Rupture; the Formation of Hæmatocele and Penetration of the Bladder.

Schwarz describes in the *Centralblatt für Gynäkologie*, 1895, No. 37, the remarkable case of a patient who, when two months pregnant, was taken with severe collapse. A tumor formed at the left of the uterus, and the patient gradually rallied from her collapsed condition. Two weeks after this she began to pass clots of blood and pieces of membrane from the blad-

der. In four weeks the tumor at the side of the uterus could not be felt, while in seven weeks the patient was convalescent, and the uterus had undergone involution.

Embryotomy in Neglected Transverse Presentations.

Mermann (*Centralblatt für Gynäkologie*, 1895, No. 36) has succeeded in performing evisceration, in cases where the fœtus was impacted in transverse presentation, by cutting through the ribs with long scissors, and emptying the chest and abdomen with the hand. The extraction of the child is usually easy after this has been accomplished.

Puerperal Tetanus Treated by Tizzoni's Antitoxin.

In the *Deut. med. Woch.*, 1895, No. 36, Walko describes a case of puerperal tetanus treated by antitoxin in von Jaksch's clinic at Prague. The treatment was unsuccessful, the patient receiving in all eighteen injections, comprising $3\frac{6}{10}$ grammes. Well-marked leukocytosis developed after the second injection. As has been shown by Kittasato, tetanus bacilli could not be recognized. The clinical picture, however, of tetanus was a perfect one.

The Treatment of Tubal Gestation by Extirpation Per Vaginam.

In the *Annales de Gynécologie*, 1895, vol. xliv., Condamin, of Lyons, describes five cases of tubal gestation treated by extirpation *per vaginam*. His method of operating was essentially that of Laroyenne, and consists in puncturing the sac and removing its contents, followed by the removal of the diseased tube and ovary. This is done by vaginal incision, followed by the insertion of an aseptic sponge to check bleeding, which is afterward replaced by a tampon of gauze. The results in the cases described were good.

The Anatomy of the Pubic Joint.

Queirel, of Marseilles, in the *Annales de Gynécologie*, 1895, vol. xliv., has examined 200 women to determine the question as to whether the pubic joint is in the median line of the pelvis or whether it is often to one side. This investigation was undertaken because, in performing symphysiotomy, operators have failed to find the joint, and have in some instances cut through bony tissue, asserting that the joint was ossified. Of these 200 women 135 had borne children, 65 had not. Their ages ranged from twenty to eighty-nine years. He found between the ages of thirty-one and forty in 39 women examined that the joint was four times upon the left of the median line and in 5 patients upon the right. In 50 women whose ages varied between twenty-one and thirty, in 7 there was a deviation to the left, in 6 to the right.

At other ages deviation was found is less frequency. In all, in the 200, in 20 per cent. the joint was not in the median line. An examination of the records of large numbers of symphysiotomies shows that the pubic joint is very rarely ossified.

Pregnancy and Labor Complicated by Ovarian Tumors.

In the *Monatsschrift für Geburtshülfe und Gynäkologie*, 1895, Band ii. Heft 3, Rubeska, of Prague, contributes an interesting paper upon this subject. He urges the importance of removing an ovarian tumor in a pregnant patient as soon as a diagnosis of its presence is made. Prompt action is especially imperative when the tumor is such as to prevent the normal progress of pregnancy. Rubeska describes the case of a patient in her third pregnancy who had an ovarian tumor. The uterus and vagina were pushed downward and the size of the tumor was such that pregnancy at five months could not proceed further. It was impossible to replace the uterus in a favorable position. Although the tumor was found to be adherent, it was successfully removed. Twenty-four days afterward the patient was spontaneously delivered.

He also removed a dermoid as large as an orange with a successful result. In this case the uterus could be pushed up above the tumor, but the latter occupied such a position that it would have impeded the delivery of the child.

A serious complication of pregnancy is found in an ovarian tumor firmly fixed by adhesions in Douglas's cul-de-sac. In such a case the writer had two courses of action from which to choose—one to remove the tumor, the other to wait until term and perform Cæsarean section. The former was chosen, and although the omentum was extensively adherent the tumor was removed. It was necessary to raise the uterus out of the pelvis and to pass a gauze drain into the vagina and replace the uterus. Thirty days after the operation the patient was delivered in spontaneous labor. Two months after the operation the patient returned with the development of a cancer in the pelvis. This diagnosis was confirmed by autopsy.

In cases where an ovarian tumor is wedged into the pelvis during labor it may be necessary to push it up, under anæsthesia, and complete the labor if possible. The writer describes a case of this sort in which the patient was brought into the hospital in labor. She afterward returned and the tumor was removed. It was found to be a dermoid, the patient subsequently perishing from the development of malignant disease of the broad ligament. Rubeska's paper emphasizes the belief held by the majority of operating obstetricians that ovarian tumors should be removed during pregnancy.

The Treatment of Abortion.

Jacub, in a recent paper before the Obstetrical Society of Moscow (*Monatsschrift für Geburtshülfe und Gynäkologie*, 1895, Band ii. Heft 3), advised that threatened abortion be treated by absolute rest, with opium or *viburnum prunifolium*. Where hemorrhage occurs without dilatation of the os and cervix the antiseptic tampon is indicated. When hemorrhage is present and a finger can be inserted the uterus should be emptied by the finger, accompanied by antiseptic irrigation. If hemorrhage is considerable and only partial dilatation is present, the finger is the safest instrument to complete dilatation. In many cases, if the patient be kept clean, the membranes and placenta will be expelled without interference and without injury. In all cases ergot should be used in daily doses during the first

week. The use of the sharp curette is not encouraged. Vaginal douches after abortion should be given for positive indications only.

Jacub calls attention to the fact that to the finger the placenta feels exactly like the endometrium, and that it is quite possible for the physician to imagine that the uterus is empty when the placenta is completely adherent. He describes two cases in which the attending physician had examined the uterus by his finger at the time of abortion, and supposed that the placenta had been removed. In both cases it was expelled several days afterward.

Two Cases of Fœtal Death During the Last Months of Pregnancy from Direct Violence.

Westphalen reports from the Obstetric Clinic of Kiel (*Monatsschrift für Geburtshülfe und Gynäkologie*, 1895, Band ii. Heft 3) the case of a patient in the latter months of pregnancy who fell upon the street and struck her abdomen against a door-step. She had severe pain for several days, followed by the cessation of fœtal movements. In two weeks afterward she was delivered of a dead child. When the placenta was expelled numerous clots came with it. On examination it was found that the umbilical vein had been ruptured and that the child had perished from hemorrhage following the accident. The cord was a short one, which undoubtedly favored the rupture.

His second case was that of a patient in the latter weeks of her sixth pregnancy who struck her abdomen against a bench while washing at a tub. Pain and cessation of fœtal movements followed. Hemorrhage gradually came on, terminated by the birth of a dead child. On examination the placenta had been loosened and the child had perished from hemorrhage.

Cæsarean Section in Uterus Didelphys.

In the *Archiv für Gynäkologie*, 1895, Band xlix. Heft 3, Tschudy reports the case of a patient in the clinic at Zurich who was brought to the hospital in labor. An examination under chloroform revealed a completely developed uterus into which the finger could be inserted, and which was found to be empty. At one side of the vagina there was a depression in the mucous membrane, which apparently marked the entrance of a second ill-developed uterus which contained the child. As the child was living, Cæsarean section was performed by Porro's method. The patient made a good recovery. Upon examination afterward the normal uterus was found in good position, while a small stump was left from the partially developed womb. The writer calls attention to a second case in which the same abnormality existed, accompanied by the presence of a vaginal septum which prevented the escape of the menstrual discharge. In this patient it was necessary to excise the septum to procure relief.

The Disinfection of the Hands.

Reinicke contributes an article upon this subject to the *Archiv für Gynäkologie*, 1895, Band xlix. Heft 3. He concludes from an elaborate series of experiments that the hands should first be cleansed with hot water and soap, and brushed for five minutes; then brushed from three to five minutes

in 90 per cent. alcohol, and afterward in an aseptic fluid. The quickest method of disinfecting the hands consists in brushing them vigorously in alcohol for five minutes.

Pregnancy Complicated by a Large Vesical Calculus.

Rosenfeld, of Nurenberg, described before the Obstetrical Society of that city (*Münchener med. Woch.*, 1895, No. 39) the case of a woman, aged twenty-seven years, brought to the clinic suffering from severe pain in the bladder, with retention of urine. She was four months pregnant. On examination a large vesical calculus was found preventing the escape of urine and complicating pregnancy. The patient was operated upon on the following day by transverse incision after Trendelenburg's method. It was impossible to distend the bladder with boric acid solution. Sutures were introduced into the bladder wall and the bladder drawn upward as far as possible and opened. The stone was so large that the incision was prolonged to the neck of the bladder. Hemorrhage was checked by a tampon. The stone was finally removed and with it a quantity of purulent urine. The bladder was cleansed and stitched to the abdominal wall and tamponed with iodoform gauze, and a drainage-tube inserted. Abortion occurred forty hours after the operation. The patient perished of pneumonia and heart-failure a few days after the operation. Peritonitis was absent. Hydronephrosis and purulent nephritis of both kidneys existed. The ureters were dilated.

GYNECOLOGY.

UNDER THE CHARGE OF

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OF NEW YORK.

Rapid Dilatation of the Unimpregnated Uterus.

Braithwaite (*British Medical Journal*, June 29, 1895) recommends the use of Hegar's dilators under anæsthesia on the last day of the menstrual period, immediately after the cessation of the discharge, when the tissues are soft and elastic. The advantages claimed for this method are the avoidance of inflammation and sepsis, of which there is considerable risk when tents are used and the rapidity with which dilatation can be effected; there is less danger of injuring the tissues, he claims, as they stretch more easily than at other times and the dilatation is more permanent.

Early Diagnosis of Cancer of the Cervix.

Sinclair (*Medical Chronicle*, 1895, No. 5) calls attention to the importance of the early recognition of cancer of the cervix uteri, and the unsatisfactory result of many microscopical examinations of bits of excised tissue. The want of a clinical sign which can be recognized by the general practitioner

has long been felt. The writer believes that such a sign is offered by the greater friability of the tissues in malignant disease. If the suspected area is exposed through a speculum and is scraped with a sharp spoon, it will simply bleed if non-malignant, but if cancerous, the soft, cheesy nature of the diseased tissue will be readily recognized.

A More Radical Method of Performing Hysterectomy for Cancer of the Uterus.

Clark (*Johns Hopkins Hospital Bulletin*, July–August, 1895), making a practical application of catheterization of the ureters, has suggested a method of extirpating a considerable proportion of the broad ligaments with the cancerous uterus. His technique is as follows: Bougies are inserted into the ureters under cocaine-anæsthesia. The patient is then etherized, a free abdominal incision is made, and the upper portions of the broad ligaments, with the ovarian ligaments, are tied. The bladder is then separated and the uterine arteries exposed and dissected out an inch beyond the vaginal branches, where they are ligated. The ureters are dissected free, when the remainder of the broad ligament is tied close to the iliac vessels and divided at its pelvic attachment, the dissection being carried well below the cancerous area. The vagina is then perforated with scissors and is tied in segments and divided. A strip of gauze is passed down into the vagina and the peritoneal flaps are sutured over the raw surface. Finally the pelvic cavity is irrigated and the abdomen closed without drainage.

Ligation of the Uterine Arteries for Uterine Fibroids.

Kuhn (*Nordiskt med. Arkiv*, August, 1895) reviews the history of the operation as suggested by Gottschalk, and mentions the results of Martin, Büstner, and Fritsch. Sixteen cases are reported from the Copenhagen clinic. In six operations by Howitz only one was successful, on account of the youth of the patients and the size of the tumors.

The Technique of Vagino-fixation of the Uterus.

Wertheim (*Centralblatt für Gynäkologie*, 1895, No. 18) reports thirty-seven operations, grouped as follows: Seven cases in which Mackenrodt's original technique was adopted, all of which were unsuccessful; nine by Dührssen's original method, with three failures; sixteen in which the vesico-uterine pouch was opened (vaginal cœliotomy), all successful; and five according to Mackenrodt's more recent method with no failures, though the intestine was punctured in one instance.

Assuming that the object of the procedure is the permanent fixation of the fundus uteri, he infers from his personal experience that the peritoneal cavity should be opened in every instance, as only in this way is the operation rendered absolutely safe. The causes of failure as maintained by Dührssen —perimetritis, metritis, and hypertrophy of the portio, the giving way of catgut sutures, and subsequent pregnancy—were noted in three of the writer's cases. The criticism that the uterus is fixed in extreme anversion does not

hold, he thinks, as the organ eventually regains its normal mobility. Vesical disturbance, especially from buried sutures, are avoided by using catgut for peritoneal fixation. In general, intra-peritoneal vaginal fixation is to be regarded as a substitute for ventro-fixation only in those cases in which the adhesions are not so firm as to prevent their separation through the vaginal incision and perfect replacement of the organ.

Ichthyol in Anal Fissure.

V. d. Willigen (*Nederl. tijdsche v. Geneeskunde; Centralblatt für Gynäkologie*, 1895, No. 18) reports several cases of anal fissure which were treated with pure ichthyol applied with a brush twice daily, and also after each defecation. The cure was rapid in every instance. The same treatment is recommended in the case of fissures of the vulva and vagina.

Hysterectomy with the Cautery.

Mackenrodt (*Centralblatt für Gynäkologie*, 1895, No. 18) presented at a meeting of the Berlin Obstetrical Society a cancerous uterus which he had removed through the abdominal incision with the Paquelin cautery, no ligatures having been used. The case was practically an inoperable one, but the operation was not difficult, and it was possible to remove more of the disease in the broad ligaments than if clamps or ligatures had been applied.

[The use of the cautery in total extirpation, though a new departure abroad, has been demonstrated by Dr. John Bryne, of Brooklyn, who has performed several successful hysterectomies in this way.—H. C. C.]

Igni-extirpation of the Uterus for Carcinoma.

Under this term Mackenrodt (*Centralblatt für Gynäkologie*, 1895, No. 24) refers to the removal of the uterus in vaginal extirpation by the cautery alone, without the use of permanent clamps or ligatures. The idea first occurred to him in connection with the prevention of primary infection during operations for malignant disease of the cervix uteri. He began by separating the cervix with the cautery, then divided the stumps in this way after previous ligation or clamping. But this did not fulfil the object, *i. e.*, to cauterize the *entire* raw surface. He accordingly determined to rely on the cautery alone for the closure of the ends of the ovarian and uterine arteries. He has performed four vaginal and two vagino-abdominal hysterectomies according to the following method: After thorough cleansing of the vagina the cancerous tissue is removed with the sharp spoon, the raw surface cauterized, and the vagina is again disinfected with soap and water, alcohol, and sublimate solution. If the introitus is narrow more room is gained by deep lateral incisions, made with the Paquelin at a white heat. The uterus is then drawn downward with a volsella, and Douglas's pouch is opened with the cautery. Then the lateral, and finally the anterior vaginal, fornices are divided in the same manner. The anterior vaginal wall is pushed upward with the thumb of the left hand, so as to put the attachments between the uterus and bladder on the stretch, which are divided by light touches with

the Paquelin. The lower portion of each broad ligament is now included in a special form of clamp, and is burned through on the uterine side; after thoroughly charring the stump the forceps are removed, care being taken not to disturb the eschars. Any bleeding point is seized with forceps and again touched with the cautery. The uterus is now drawn downward still more, and the separation of the bladder and vesico-uterine fold of peritoneum is completed with the cautery. Under the guidance of the fingers the upper portions of the broad ligaments are grasped in turn with the temporary clamps, and are separated as before, the intestines being meanwhile protected with gauze. Great care is necessary in cauterizing the stumps containing the ovarian arteries, and sometimes it may seem advisable to apply ligatures, although the writer has been obliged to do this in only one instance. The wound is dressed in the usual manner. When there are secondary deposits in the upper portions of the broad ligaments, the writer secures the uterine arteries from below, and when it is evident that firm adhesions are present he opens the abdomen and completes the operation from above. The four cases of vaginal hysterectomy presented no difficulties whatever, and were all successful. One death followed the two combined operations, the patient succumbing on the eighth day from exhaustion due to previous hemorrhages. The operations all having been performed since February of this year, sufficient time has not elapsed to allow a comparison between this and the usual method as regards freedom from recurrence. The fact that the vessels could be thoroughly secured by cauterization was fully demonstrated, and the advantage of this method over the use of clamps was shown by the absence of pain and other disturbances which so often accompany the application of the latter.

The Avoidance of Infection in Hysterectomy with the Thermo-cautery.

Rosner (*Centralblatt für Gynäkologie*, 1895, No. 21) calls attention to the well-established fact that direct infection of healthy wounds can occur during the removal of malignant growths, especially in vaginal extirpation of the uterus for carcinoma of the cervix, which cannot be prevented by either previous cauterization of the diseased surface or by constant irrigation during the operation. He recommends the entire removal of the uterus with the thermo-cautery (thermo-cauterectomy), and reports four successful cases. The technique is as follows: The uterus being drawn forcibly downward, the diseased surface is thoroughly cauterized; then the cervix is freed and the bladder dissected off with the fingers. Douglas's pouch is opened with the cautery, the bladder is held upward, the uterine arteries clamped, and the lower portion of the broad ligaments are burned through. The diseased cervix is now amputated as high as possible with the cautery, the remainder of the organ is drawn downward, the upper portions of the broad ligaments clamped and separated as before, and the uterus and adnexa are removed. The stumps are covered with iodoform powder, and the usual gauze dressing is applied.

[This operation can in no sense be regarded as hysterectomy with the cautery alone; it is the ordinary clamp-operation in which the cautery is

used simply as a substitute for the scissors in dividing the broad ligaments.—H. C. C.]

New Operation for Retro-displacement.

Czempin (*Ibid.*) reports thirty cases of operation for movable retroflexion with only two failures. His method is especially adapted, he states, to displacements occurring in virgins who cannot wear pessaries. After curettage a special form of sound is introduced into the uterine cavity, the organ is anteflexed, and the patient is placed in Trendelenburg's posture. A median incision two inches in length is made just above the symphysis, and the peritoneum is exposed and dissected off for a short distance around the edges of the wound, but is not opened. The fundus is pushed upward in contact with the peritoneum so that no intestine shall be in the way, and is secured by silk sutures passed through the edges of the wound and the opposed parietal and visceral peritoneum. After closing the fascia with catgut and the skin with another layer of the same sutures, the fixation-sutures are tied over a roll of gauze and are left in position for two weeks.

[This is simply the so-called "transperitoneal hysterorrhaphy," which was described by Krug, of New York, several years ago.—H. C. C.]

The Influence of Castration on Metabolism.

Curatulo and Tarulli (*Centralblatt für Gynäkologie*, 1895, No. 21), as the result of experiments upon animals, arrive at the following conclusions: The ovaries, like other glands, have a secretion peculiar to themselves, which is separated from the blood, but the chemical composition of which is at present unknown. It possesses the property of oxidizing phosphorus compounds, or those which furnish the lime-salts of the bones. Hence after the removal of the ovaries in cases of osteomalacia there is an increased deposit of calcium and magnesium phosphates in the bones, thus restoring the normal firmness of the latter.

The increased deposit of fat observed after the normal and artificial climacteric may be explained by reference to the diminished oxidation of fat through the absence of the same secretion.

Freund's Operation for Prolapsus Uteri.

Gaertig (*Centralblatt für Gynäkologie*, 1895, No. 22), in reporting seven operations for prolapsus by Freund's method performed by Brunn, calls attention to the fact that the procedure is not a new one, a case having been reported by Schramm, who referred to it as simply the revival of an old operation. The writer is not very enthusiastic in its favor, as only two patients remained cured at the expiration of a year. The technique is extremely simple, and no bad results followed in any instance, except transient œdema. While the simplicity and harmlessness of the operation must be admitted, one should be careful not to promise a permanent cure.

Adhesive Peritonitis Following Cœliotomy.

V. Mangoldt (*Ibid.*) reports a case of castration and ventro-fixation in which the patient, having been well for five months after the operation, began to suffer from severe abdominal pains, obstinate constipation, and difficult urination, the cause of which could not be determined. On opening the abdomen adhesions were found between the omentum and the stumps, while a space existed in front of the fundus uteri and the broad ligaments in which intestine might become incarcerated. The adhesions were separated and the partly-detached fundus was again sutured to the anterior abdominal wall.

For two years the patient enjoyed good health; then she began to have violent colicky pains, especially in the region of the cicatrix, and occasional vomiting; emaciated rapidly. A third cœliotomy revealed an adhesion of the omentum along the line of the old cicatrix, as well as to the fundus uteri. The omentum was separated, ligated, and excised, the patient making a rapid afebrile recovery, with entire relief from her former pains. Soon after she suddenly developed symptoms of ileus, for which it was necessary to open the abdomen a fourth time, when an adhesion was found between the stump of the omentum and the cicatrix, under which a coil of intestine was imprisoned. Other parietal and visceral intestinal adhesions were found and separated. The patient again recovered rapidly, and had gained twenty pounds since the last operation.

All four cœliotomies were strictly aseptic, no fluid was introduced into the abdominal cavity, adhesions being separated by scissors, the Paquelin, or by blunt dissection. The bowels were moved on the third day after each operation, and were kept open afterward, yet the adhesions formed nevertheless.

The Anatomy of Carcinoma Uteri.

Veit (*Centralblatt für Gynäkologie*, 1895, No. 24) makes some important practical suggestions growing out of his more recent anatomical studies of this subject. He does not think that sufficient attention has been paid to the different varieties of cancer in the giving of a prognosis as regards recurrence after hysterectomy, nor is he inclined to agree with Winter in his statement that a considerable proportion of such recurrences are due to the infection of healthy raw surfaces during the operations. He divides cases of carcinoma of the cervix clinically into those in which the disease is situated in the portio or in the cervical canal, and in which it appears in a nodular form in the cervix, or, as disease of the corporeal endometrium. Primary nodules in the cervix originate in the connective-tissue, and are now regarded as endothelioma. As regards extension of the disease, the writer has often found cancer-cells in the lymphatics of the broad ligaments; when these infected vessels are divided during operation it is natural to infer that they form foci, from which a rapid recurrence takes place. The latter form of recurrence is characterized by the fact that the metastatic deposits present the same histological structure as the primary disease, and that they rapidly invade the deeper tissues. The simultaneous appearance of two apparently independent primary cancerous nodules can usually be explained by referring the more deeply-seated one to secondary infection.

PÆDIATRICS.

UNDER THE CHARGE OF

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FRESH-AIR TREATMENT OF PERTUSSIS.

ULLMANN (*Jahrbuch f. Kinderheilkunde*, Bd. xl. S. 39) is thoroughly convinced of the efficacy of the fresh-air treatment of pertussis. For sake of comparison between the period passed out of doors and that passed in the house, he has noted, in a series of cases, the hours spent in each, and the number of paroxysms and their intervals under the two conditions. From these data he concludes that the fresh-air treatment is one of the most efficacious, if not the only one, for the management of a case of whooping-cough. The child should pass the entire day out of doors, not only in the warm season, but even at all times of the year, provided it be not stormy. It is considered only necessary to prevent the child from running or talking or otherwise provoking an access of coughing. Contrary to general teaching, he advocates the open-air exposure of the patients even when there is bronchitis or broncho-pneumonia, whether consecutive to pertussis or not, the only precaution being to have the little ones well wrapped up. This plan he has practised with infants a year old suffering from grave broncho-pneumonia. During this treatment the author employs neither a specific nor narcotics. In the presence of very grave broncho-pneumonia he prescribes spirits of camphor with an expectorant.

THE IMMUNIZING EFFECTS OF ANTITOXIN.

MORRILL (*Boston Medical and Surgical Journal*, June 27, 1895) reports the results of an extensive experience in the use of antitoxin as an immunizant in the wards of the Boston Children's Hospital. A total of 438 injections were given between January 13th and May 13th—of these 109 were of Gibier's serum, 104 of Behring's, 74 of Aronson's, and 151 of the antitoxin prepared by the Massachusetts State Board of Health. The largest number administered to one child was 7. Many of these patients were under treatment for acute diseases at the time of injection—croupous and broncho-pneumonia, grippe, tubercular peritonitis, empyema, typhoid, and various surgical conditions. Dangerous symptoms were observed in only one case: a boy, aged two years, with leucocytosis and enlarged spleen, upon whom two preceding doses had produced no effect, had a temperature of 105.4° F., and considerable œdema about the point of injection. These symptoms promptly subsided after stimulants and cold baths. As a rule, the temperature was

observed to rise less than a degree a few hours after injection, and in 75 per cent. of all cases this was all there was to be seen. Erythema around the point of injection occurred in 23 cases. In 4 it was very extensive, and closely resembled erysipelas. The average duration of this symptom was three days; in some cases it lasted only a few hours. It usually appeared on the first day after antitoxin, and never later than the eighth. Urticaria occurred 28 times. The frequency, severity, time of appearance, and duration of this symptom varied greatly with the brand of serum employed. That of Gibier (Pasteur Institute of New York) produced it in 22 per cent. on the (average) seventh day, and lasting (an average of) two and a half days. Behring's caused it in but 1 case, appearing on the eighth day and lasting three days. Aronson's gave rise to no urticaria. The serum of the Massachusetts State Board of Health produced in about 4½ per cent. an eruption, appearing on the (average) second day, and disappearing in a day and a half. This serum at present very rarely gives rise to anything cutaneous, although at first urticaria was of pretty common occurrence. In three instances the erythema was papular and very closely resembled measles. In two instances an eruption like scarlet fever was present. Occasionally a child whose skin remained clear after the first or second or third immunization, broke out with an erythema or an urticaria after a subsequent injection. The same brand of serum may produce an urticaria, an erythema, or no skin eruption whatever in the same subject, as proved in more than one instance where repeated injections were given. Pain (at times intense) was noticed about the point of injection in 16; œdema in 7; axillary or inguinal glands enlarged in 3; general malaise (in 1 instance a mild delirium) in 3. Pain in the articulations in less than 1 per cent. Nausea, with or without vomiting, in 2 per cent. Diarrhœa (ephemeral) in less than 3 per cent. Increased frequency of micturition was noted in rare instances. The urine of 82 children was very carefully examined with reference to the effects of antitoxin on the kidneys, and the result of 540 examinations may be briefly stated as follows: In 20 per cent. no change whatever was detected. Of the remaining 80 per per cent., those which had been free from albumin previous to immunization, showed a slight trace—in most instances the slightest possible amount, which could be detected only when placed in front of a dark background. This within twenty-four or forty-eight hours after injection. In cases where albumin had been present before the use of antitoxin, there was a very slight increase of the amount. In no case was there any diminution in the amount passed, or any evidence of failure to eliminate properly. Repeated microscopic examinations revealed nothing more than an insignificant amount of renal irritation in any case; and when the albuminuria was produced by antitoxin, it disappeared in a period varying from two to three days to a fortnight.

With regard to the protection afforded by antitoxin, his experience has tended to show that the serum, when fresh, can be relied upon to immunize against anything resembling clinical diphtheria for thirteen days, and very probably for a longer space of time. Moreover, in no instance was the bacillus detected in the nose or throat of any child who started with a clear record in a shorter space of time than that just mentioned. This last point (if eventually established) is of practical importance as showing that an immunized

child is not only safe from diphtheria, but is also safe as concerns others for a definite length of time.

Diphtheritic Lesions of the Nervous System.

Crocq, fils (*Congress of Internal Medicine*, Bordeaux, 1895; *Revue Mensuelle des Maladies de l'Enfance*, September, 1895) reports some interesting experimental researches upon the nerve-lesions of diphtheria. Rabbits were inoculated with four-months-old culture of Lœffler bacilli, weak enough to intoxicate but to assure life for periods varying from one to three months. Disturbances of motion were invariably observed, but sensation was affected in but one case. Autopsies showed that the diphtheritic poison acts upon the cord, the nerves, rarely upon the medulla, and never upon the brain. These lesions consist of swelling of the nerve-cells, loss of their protoplasmic prolongations, disappearance of nuclei and extensive proliferation of the cells of the ependyma. Acute myelitis is rare. The toxine seems to have an elective action upon the nerve-cells, and only exceptionally determines proliferation of the connective tissue. The lesions of the nerves appear from the fifth day; the axis-cylinder segments, the protoplasm becomes more abundant, the myeline becomes granular and is absorbed, and the axis-cylinder disappears. The anterior nerve-roots are always affected, but the posterior only show some degenerated fibres arising from cells of the anterior horns. The rare lesions of the inferior portion of the medulla do not progress, and may even disappear. In the rabbit it seems that the cord lesions precede those of the nerves. Whether this occurs in man is an open question. With him diphtheritic paralyses are of two kinds—one localizes itself in the bucco-pharyngo-laryngeal apparatus, the other is generalized. Experiment, however, produces only the latter class, dependent upon lesions of the cord. It seems, therefore, that the generalized paralyses observed in man are of spinal origin, and that the nerves are altered secondarily, while the localized forms affecting certain fasciculi and accompanied by anæsthesia are due to primary neuritis.

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CONTENTS.

ORIGINAL COMMUNICATIONS.

	PAGE
On the Visceral Complications of Erythema Exudativum Multiforme. By William Osler, M.D., F.R.C.P. Lond.	629
Nephrectomy for Cystic Adenoma in a Pregnant Woman—Recovery. By Charles L. Scudder, M.D.	646
Infantile Scurvy from Sterilized Milk. By Louis Starr, M.D.	652
Craniotomy for Epilepsy and Allied Conditions. By R. J. Hall, M.D.	655
Chronic Dyspepsia in Children. By J. Walter Carr, M.D., M.R.C.P., Lond.	666
Traumatic Enophthalmos, with a Case. By G. E. de Schweinitz, M.D.	673
A Case of Dermoid Tumor of Both Ovaries Complicated by a Deposit of Bone Upon Each Side of the True Pelvis, having no connection with the Tumors. By Charles P. Noble, M.D., and Joseph P. Tunis, M.D.	677

REVIEWS.

Cystitis and Infection of the Urinary Tract. By Dr. Max Melchior	683
A Text-book on Nervous Diseases, by American Authors. Edited by Francis X. Dercum, A.M., M.D., Ph.D.	684
Physical and Natural Therapeutics. By Georges Hayem, M.D. Edited by Hobart Amory Hare, M.D.	691
A Treatise on the Nervous Diseases of Children, for Physicians and Students. By B. Sachs, M.D.	692
A Handbook of Medical Diagnosis for Students. By James B. Herrick, A.B., M.D.	695
Clinical Lectures and Essays on Rickets, Tuberculosis, Abdominal Tumors, and Other Subjects. By Sir William Jenner, Bart., G.C.B.	696

PROGRESS OF MEDICAL SCIENCE.

THERAPEUTICS

	PAGE		PAGE
Cycling and Heart Disease	697	The Use of Oxygen Gas with Ether for Anæsthesia	700
Antagonism Between Morphine and Cocaine	698	Treatment of Diabetes Mellitus	701
Solanum Carolinense	698	Treatment of Morbus Cordis	701
Temperature in Chloroform Narcosis	698	The Reduction of Oxyhæmoglobin in Living Tissues	702
Mescal Buttons	699	Diphtheria Antitoxin	702
Apocynum Cannabinum	700		

MEDICINE.

Gastrolith	703	Acute Leukæmia	706
The Transmission of Variola	704	Cardiac Arhythmia in Childhood	707
Symptomatology of Cerebellar Disease	704	Chronic Coffee Intoxication	707
Necrosis of the Mandible in Tabes	705	Salt Solution Injections in Heart Failure during Chloroform Narcosis	708
Intermittent Pneumonia	705	The Gastric Juice in Hanot's Disease	708
Disseminated Lesions in Lead-poisoning	705	Gonorrhœal Pleurisy	708
Excessive Intestinal Putrefaction	706		

SURGERY.

Hydrotherapy in Appendicitis	709	Superficial Syphilitic Adhesion	710
The Question of Catgut Infection	709	Umbilical Hernia of the Newborn	710
Septic Infection within the Cranium	709	Cure of Superficial Varicose Veins	711

	PAGE		PAGE
Splenectomy for Echinococcus of Spleen	712	Excision of Gasserian Ganglion	713
Genital Hyperæsthesia Associated with Shortness of the Frenum Glandis	713	A Study of Lymphangitis of Ano-rectal Origin	713

OTOLOGY.

Teeth and Ears	715	Ear-massage	716
Otic Facial Paralysis	715	Chronic Purulent Otitis Media	716
Acute Otitis Media in Pneumonia	715	Septic Otitic Sinus phlebitis	717
Drainage in Acute Otitis Media	716	Cerebellar Abscess	717

DERMATOLOGY.

Treatment of Diseases of the Skin	718	Massage in Treatment of Acne of Face	720
Autointoxication and Skin Diseases	719	The Physiology of the Trichophytons	720
Dermatosis of Duhring in Pregnancy	719	Transformation of One Variety of Cutaneous Tuberculosis into Another	720
Elements of Molluscum Contagiosum	719		

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

Mucous Polypi of the Nose	721	Chancre of the Tonsil	723
Chronic Disease of the Ethmoid Cells	722	Inhalation	724

OPHTHALMOLOGY.

Bilateral Ophthalmoplegia Externa	724	Field of Vision in "Neglected Eyes"	725
Which to Slit for Probing Nasal Duct?	725	Real Extent of the Color Fields	726
Perforating Injuries of the Eye	725	Scleral Puncture	726

OBSTETRICS.

Ovarian Tumors	727	Aluminium Case for Instruments	729
Puerperal Bradycardia	728	Obstetric Study by Frozen Sections	729
Diphtheria and Puerperal Sepsis	728	Symphysiotomy with Osteoplastic Restoration of the Symphysis	730
Development of the Fœtal Cranium	729	Spontaneous Evolution during Labor	730
Scorbutus from Sterilized Milk	729		

GYNECOLOGY.

Operation for Cystocele	731	Adhesion of Appendix to Pelvic Organs	733
Treatment of Vesico-vaginal Fistula	731	Tuberculosis and Carcinoma of Uterus	733
Micro-organisms in Pyosalpinx	732	Steam as a Hæmostatic	733
Vaginal Hysterectomy with Clamps	732	Sterilization of the Vagina	734
Relation of Hæmatemesis to Menstruation	732	Salipyrin in Uterine Hemorrhage	734
Œdema of the Bladder	732	Ill Results of Pelvic Massage	734
Chronic Fibrous Peritonitis	733	Sea-bathing and Menstruation	734
		Ultimate Results of Ventro-fixation	735

PÆDIATRICS.

Morbid Anatomy of Infantile Paralysis	735	Brachial Diplegia from Syphilis	736
Use of Diphtheria Antitoxin	736	Elimination of Lime Salts	737

PATHOLOGY AND BACTERIOLOGY.

Pus	738	Syphilis and Tuberculosis	740

HYGIENE AND PUBLIC HEALTH.

Schools and Diphtheria	741	Dry Methods of Dealing with Urine	744
Water Filtration at Berlin	743		